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THE HISTORY AND DISTRIBUTION OF *PHOMOPSIS PSEUDOTSUGAE* IN EUROPE[1]

MALCOLM WILSON AND GLENN GARDNER HAHN

INTRODUCTION

A very important disease, primarily of the Douglas fir, *Pseudotsuga taxifolia* (Lam.) Britt.,[2] a conifer which, since the World War, has been extensively planted in Great Britain, is a canker (Fig. 1) and die-back attributed to *Phomopsis pseudotsugae* Wilson (42). The canker is essentially a disease of young trees. The die-back of the tops and branches, which also occurs on larger and mature individuals, may become particularly serious in young plantations of the green form (*Pseudotsuga douglasii* Carr.).[2] The latter type of disease, when occurring in connection with stem canker on trees approximately less than eight feet in height, may result not only in a much reduced final stocking, so far as the mature stand is considered, but economic loss, as well, by the death of trees which otherwise might have been of value as thinnings.

As yet, the Douglas fir canker fungus, described and figured by Wilson (40, 41, 42) as the Phomopsis disease of Douglas fir and other conifers, is not known in North America. In the light of our experience of the highly dangerous nature of imported parasites as the result of their introduction into a new environment with a consequent change in their virulence and life habits, we are forced to recognize in the Douglas fir canker of Great Britain a potential danger to the Douglas fir in North America. Considering the value of this tree in the United States from the standpoint of merchant-

[1] Paper by Dr. Malcolm Wilson, Mycological Department, University of Edinburgh, at the Royal Botanic Garden, Edinburgh, and Glenn Gardner Hahn, Office of Forest Pathology, Bureau of Plant Industry, U. S. Department of Agriculture.

[2] In the United States only one species of Douglas fir, *Pseudotsuga taxifolia*, is recognized. In Europe this American species is considered to include two distinct species —*P. douglasii*, (commonly called coast, Pacific coast, Oregon, or green Douglas fir), and *P. glauca* Mayr. (commonly called mountain, Rocky Mountain, Colorado, or blue Douglas fir). *P. douglasii* var. *caesia* Schwer. is regarded as the Fraser River form. This name has been pointed out by Boyce (3) as a misnomer. Biological differences tend to support the European species concept of Douglas fir. These differences have been indicated by Boyce (3) as the result of his observations of the diseases of Douglas fir in Great Britain and Denmark. For convenience, in this paper, the European usage is followed.

FIG. 1. Typical canker with the fruiting bodies of *Phomopsis pseudotsugae* on the green form of Douglas fir (*Pseudotsuga douglassi*) collected by Perley Spaulding at Prestwick, Ayrshire, Scotland. May, 1923. Approx. $\frac{3}{4}$ nat. size.

able timber and as a forest crop of the future, any disease, which may be looked upon as a menace to the species, should be regarded with close scrutiny and suspicion.

On account of the great importance of the Douglas fir canker, the junior author has been working in cooperation with Dr. Malcolm Wilson in further investigating the disease to prove that it is actually caused by *Phomopsis pseudotsugae*, and to discover the method of infection, the origin of the disease, its original host, its present distribution, and its relation to somewhat similar diseases which have already figured in European mycological and forestry literature. The last three subjects will be discussed in this paper.

IMPORTANT EUROPEAN DISEASES OF CONIFERS RESEMBLING THE DOUGLAS FIR CANKER

Wilson (42) and Boyce (3) have already discussed the close relationships and the large amount of confusion which has accumulated in the literature for approximately the last half century, concerning the identity of *Phoma pitya* Sacc. (32, p. 126) and *P. abietina* Hartig (11, p. 124–126), both attributed parasites of the Douglas fir and other conifers. Since the fairly recent description of *Phomopsis pseudotsugae* by Wilson in 1920 (41, 42), this fungus has been regarded by some investigators as synonymous with the older fungi originally described by Saccardo and Hartig. It therefore became highly necessary from a practical standpoint to differentiate the newly described organisms from the older and well-known parasites which may have been introduced into the United States in previous years on ornamental or plantation stock of various conifers.

In 1885, Rostrup reported *Phoma pitya* as a parasite of plantation stock of Norway spruce (29, p. 10) and later recorded it on other conifers in Denmark (30, 31). Since this time confusion has been very evident. The Danish mycologist regarded his organisms as synonymous with the classical fungus *P. abietina* which is attributed as the cause of the "Einschnürungskrankheit" of the silver fir. Following Rostrup, diseases of the Douglas fir generally described as a girdling or die-back of the shoots were reported by Somerville (34) in Scotland (1898), by Lindau (20) in Germany (1908), by Schwappach (33) in North Germany (1909), by Visart and Bommer (39) in Belgium (1908), by Farquharson (7) in Scotland (1911), by De Koning (18, p. 160) in Holland (1922), by Neger (25, p. 173) in Germany (1924), and by Fabricius (6) in Denmark (1926). These authors all identified the organism associated with the diseases as *P. pitya*, a fungus on Scots pine, *Pinus sylvestris* L., which had been briefly described by Saccardo.

Rostrup and Schwappach regarded *Phoma pitya* as identical with Hartig's fungus, *Phoma abietina,* on the silver fir. Other European investigators, however, regarded the latter fungus as a distinct species. It was reported by Böhm (2) in 1896 and Hausrath (12) in 1923 on the Douglas fir in Germany. In France (1890) Hartig's fungus was regarded as *Fusicoccum abietinum* (Hart.) Prill. et Del. by Prillieux and Delacroix (28), and Maublanc (22). Mer (23, 24) published extensively on the disease with regard to its pathological symptoms and histology and made, incidentally, a brief reference to his inoculation experiments. This investigator, together with Henry (13), seemed to prefer to call the fungus by its original name *Phoma abietina.* In recent years *P. abietina* has been reported by Doidge (5) from South Africa (1924) and Stevenson (35, p. 3) from the United States (1926). Considerable uncertainty, however, appears to have existed in the minds of European mycologists over the exact identity of *P. pitya* and *P. abietina.* This uncertainty was expressed in 1897 by Tubeuf and Smith (38, p. 465). Tubeuf (37), in his text book, had drawn attention to Rostrup's description of *P. pitya* quoted above and mentioned that it closely resembled *P. abietina.* Smith, in a footnote in the English translation, suggested that there had been some confusion between the two species. This confusion, no doubt, was largely due to the scant description of the former fungus. The organism recognized in different parts of Europe as *P. pitya* and regarded as being identical with Saccardo's original fungus collected by Magnus apparently held certain marked morphological resemblances to Hartig's fungus. Further, the pathological host symptoms of Hartig's fungus appeared to be identical with those attributed to the older fungus *P. pitya.* Although Hartig described this fungus only on *Abies pectinata* DC., it was also regarded by investigators as occurring on other conifers, including Douglas fir. In 1913 Lind (19), in going over Rostrup's herbarium, made the new combination of *Phomopsis pitya* (Sacc.) Lind based on the collections of *P. pitya* identified by Rostrup; he regarded Rostrup's fungus as identical with the *P. abietina* of Hartig.

In 1918 Grove (8), in discussing the identity of *P. pitya,* stated that Saccardo's fungus was undoubtedly *Sclerophoma pitya* (Sacc.) Died. and that possibly "*Phomopsis pitya,* Lind = *Fusicoccum abietinum* Prill. et Delacr." might be synonymous. Three years later (9) he made the combination *Phomopsis abietina* (Hart.) Grove, including *Phoma abietina* Hart. and *Phomopsis pitya* Lind as synonyms. His description of the fungus, on which this combination was based, was apparently not derived from a study of authentic material on the silver fir from either Germany or France, where Hartig, and Prillieux and Delacroix, respectively, had described the organism, but from diseased specimens of Douglas fir from

Scotland. His description of this Douglas fir Phomopsis differs considerably from that given by the German and French authors of the continental fungus on *Abies pectinata.*

Hickel (14), in his monograph of the Douglas fir (1922), mentions the diseases of that species in Europe. He lists *Phoma pitya, P. abietina,* and *Phomopsis pseudotsugae* as synonymous. Jørstad (17, p. 169–170) in 1925 reports that while he has not seen Norwegian material of *P. abietina* on Abies, the *P. pitya* destroying the tops of silver firs at Sand in Ryfylke, recorded by Schøyen, may be *Phomopsis abietina* (Hart.) Grove. He further states, "*Phomopsis pseudotsugae,* which is known as producing similar girdling disease primarily on *Pseudotsuga douglasii,*" has not been recorded from Norway, although a twig disease resembling it has been observed.

A perusal of the foregoing discussion clearly demonstrates the confusion in the literature relative to the exact identity of the three organisms to which similar girdling and die-back diseases of the Douglas fir and other conifers have been attributed. The solving of this problem was of particular importance to North America because of the acute necessity for complete recognition of the European parasite *Phomopsis pseudotsugae* on conifers in the United States and Canada.

THE IDENTITY OF PHOMA PITYA

For a full discussion of the identity of *Phoma pitya* and *P. abietina* and their relationships to *Phomopsis pseudotsugae* the reader is referred to a recent paper published by the authors (44). As a result of the examination of the type specimen of *P. pitya,* the fungus was found to be neither a Phoma nor a Phomopsis but a Sclerophoma. Accordingly, the fungus has been described as *Scl. magnusiana* Wilson et Hahn in honor of Professor Magnus, its discoverer in 1878. The following synonyms are given: *Phoma pitya* Sacc., *Scl. pitya* (Sacc.) Died., non *Scl. pitya* (Thüm.) v. Höhn.

Sclerophoma magnusiana produces its spores, so far as could be ascertained, by a budding process from the hymenial cells lining the spore cavity. No evidence of endogenous spore origin as reported by von Höhnel (15) for this genus could be observed. Van Luyk (21) describes a similar budding process for spore formation in *Scl. pityophila* (Cda.) v. Höhn., and Archer (1, p. 11) reports the same for *Scl. concaviuscula* (E. et E.) v. Höhn, *Dothichiza minor* (E. et E.) v. Höhn, and *Scarcophoma miribelii* (Fr.) v. Höhn. Archer has stated that it is to be regretted that certain genera drawn from the genus Phoma by von Höhnel and placed in the Endogenosporae can not be retained in their original form as erected by him. A further critical morphological study, together with a cultural

study of the various species of Sclerophoma (with special reference to those occurring on confers), is necessary to supplement observations already made by von Höhnel (15, 16), van Luyk (21), Petrak (26, 27), and Archer (1). The validity of the genus Sclerophoma is still somewhat uncertain and until further investigation has been undertaken the assignment of *Scl. magnusiana* to it must be regarded as provisional.

Sclerophoma magnusiana is known to occur on Scots pine as a saprophyte. While certain of the other species of Sclerophoma reported on conifers may be suspected of being weakly parasitic, there have been no observations made which would suggest that *Scl. magnusiana* is anything but a saprophyte.

THE IDENTITY OF ROSTRUP'S SPECIMENS OF PHOMA PITYA

The fungi which Rostrup recognized as parasites on the terminal shoots and branches of the Douglas fir and other conifers and which he identified as "*Phoma pitya* Sacc. = *P. abietina* Hartig," have been examined and the details of this examination have been recently published (10) by the junior author. It was found that Rostrup had included a number of fungi under this identification. These fungi included *Phomopsis strobi* Syd., *P. pseudotsugae*, (?) *P. juniperovora* Hahn, a *Phomopsis* sp. and *Sclerophoma* spp., including very probably *Scl. pityophila* (Cda.) v. Höhn, all of which Lind (19) called *Phomopsis pitya* (Sacc.) Lind. The *Phomopsis* sp. included in the above collections has been identified with a fungus originally described on cones as *P. conorum* (Sacc.) Died. (*Phomopsis pitya* Lind, pro parte). The emended description of the fungus is based on freshly collected Scottish material occurring on the Douglas fir, which is morphologically identical with the Danish specimens collected by Rostrup. *P. conorum* is regarded as a saprophyte, probably becoming only weakly parasitic under certain host conditions. Hahn (10) frequently observed the organism in connection with frost injury amongst nursery and plantation stock of Douglas fir associated with the killing back of the terminal shoots, which closely resembled the die-back on the same host caused by *P. pseudotsugae* (Fig. 2, C). *P. conorum* has never been observed associated with body cankers (Fig. 2, D) on the trunks and larger branches of the Douglas fir.

THE IDENTITY OF PHOMA ABIETINA

The actual type specimens of this fungus, identified by Hartig as *Phoma abietina* and by Prillieux and Delacroix as *Fusicoccum abietinum* (Hart.) Prill. et Del., were not examined, but authentic material actually collected and identified by the German author and by Magnus, and field material from the Jura (Fig. 2, A) freshly collected and received through the cour-

tesy of Professor Guimer, of the College of Forestry, Nancy (1926), were available for critical study.

The above-mentioned material answered exactly the description of the fungus given by the German (11) and French authors (28). The writers are, however, inclined to regard the fungus as a Phomopsis and have described it as such (44), giving it the new combination *Phomopsis abietina* (Hart.) Wilson et Hahn with the following synonyms: *Phoma abietina* Hart.; *Dothiorella pitya* Prill. et Del., nec Sacc.; *Fusicoccum abietinum* (Hart.) Prill. et Del.; nec *Phomopsis abietina* (Hart.) Grove. As has been pointed out above, Grove (9) in making his new combination, based his description not upon authentic material collected on the silver fir in France or Germany, but on specimens collected on the Douglas fir in Scotland. The morphological characters given by him in the description showed a marked difference from those given by Hartig and the French authors in their original descriptions.

The only typical material of *Phomopsis abietina* seen by the authors has been collected in Germany and France. The fungus has not been observed so far on Abies or on Douglas fir in Great Britain, although the organism may possibly be present in that country. The authors would be somewhat inclined to regard the fungus as a continental species, not coming normally so far north as the British Isles. Jørstad (17, p. 169) reports that he has never observed the organism in Norway. Professor Ferdinandsen and Magister Rostrup of the Plantepatologisk Laboratorium at Copenhagen, in correspondence with the junior author, state that they have not observed the typical symptomatology of *P. abietina* on the silver fir in Denmark.

Phomopsis abietina is the attributed cause of the classical "Einschnürungskrankheit" (Fig. 2, A) figured and described in detail by Hartig (11) and Mer (23, 24). Considerable controversy has occurred in France concerning the seriousness of the disease. In certain years the fungus seems to have strongly manifested itself in France and in Germany associated with a girdling of the smaller branches and stems of the silver fir. Large diameters were not attacked and trees were never killed outright by the disease. Today the disease on the Continent, while recognizable, is not regarded as a particularly serious one.

Whether *Phomopsis abietina* occurs on the Douglas fir on the Continent is problematical. Böhm (2), Schwappach (33), and Hausrath (12) have all reported it on this host in Germany. It is very possible that they might have confused other Phomopsis species, e.g., *P. pseudotsugae* or *P. conorum*, with this fungus. As Boyce (3, p. 4) has pointed out, Böhm's (2) description of the effect of the fungus on the host strongly suggests *P. pseudotsugae*.

FIG. 2. A. Girdling or "Einschnürungskrankheit" of a smaller branch of silver fir (*Abies pectinata*) attributed to *Phomopsis abietina*. Freshly collected material from the Jura, France. Natural size. B. Girdling of a four-year-old tree of Douglas fir caused by *Phomopsis pseudotsugae*. Natural size. C. Die-back of the leading shoot of a four-year-old tree caused by *Phomopsis pseudotsugae*. Practically the entire terminal growth is dead. ½ natural size. D. The canker has killed an eight-year-old plantation tree of Douglas fir by practically girdling the trunk. The dead cortical tissues are scaling off in the characteristic plate-like manner. ¼ natural size.

Specimens reported by Doidge (5) of *Phomopsis abietina* from South Africa have not been examined. The specimen of this fungus cited by Stevenson (35) in the United States on *Abies veitchii* has been examined through the courtesy of Dr. H. C. Young, Ohio Agricultural Experiment Station, and was found not to be *P. abietina*. The identity of this fungus will be discussed in a later paper.[3]

PHOMOPSIS PSEUDOTSUGAE, A PARASITE OF THE DOUGLAS FIR

A consideration of the foregoing, together with the descriptions of the organisms, *Sclerophoma magnusiana* (*Phoma pitya*), *Phomopsis abietina* (*Phoma abietina*), and *Phomopsis pseudotsugae*, recently published (44), indicates the separate identity of the three coniferous fungi.

Sclerophoma magnusiana, so far as observations have been made, is not associated with a girdling of the stems of Scots pine. The *Phoma pitya* reported by Neger (25, p. 173) on Douglas fir and *Pinus strobus* as causing an *Einschnürungskrankheit* may have been a Phomopsis, possibly *P. pseudotsugae*. Sydow (36) has described *P. strobi* on white (Weymouth) pine from North America but he does not mention an attendant girdled condition of the host.

Phomopsis abietina, on the other hand, is a species associated with a typical *Einschnürungskrankheit* (Fig. 2, A) but the organism has never been actually shown to be a definite parasite. The fungus has not been observed occurring on cankers on the larger diameters and trunks. Only the smaller twigs and branches are girdled and, as a result, the parts beyond are slowly killed, becoming reddish brown and showing strikingly amongst the healthy green foliage. Cultural studies of this fungus from authentic material have been made and these have shown distinct differences from *P. pseudotsugae*. These cultural distinctions, in addition to differences in morphology, entirely dissipate all former confusion attendant on the identity of the two Phomopsis species.

Phomopsis pseudotsugae may be regarded as a true parasite. Experiments[4] performed under natural conditions have given positive results. The Douglas fir parasite causes three types of lesions,—first, a definite die-back of the young branches and shoots (Fig. 2, C); second, a typical girdling (Fig. 2, B) wherein the death of the tissues beyond the girdled area takes place much more slowly than the tissues involved in the preceding die-back; and third, a definite canker (Fig. 1; Fig. 2, D) of limited growth

[3] Hahn, G. G. Life-history studies of the species of Phomopsis occurring on conifers. (Unpublished manuscript.)

[4] The results of these experiments conducted by the junior author in cooperation with Dr. Wilson will be published shortly. (Parasitism of *Phomopsis pseudotsugae*, unpublished manuscript.)

on the larger branches and trunks, which normally develops during the winter and spring following infection and stops completely when the host resumes active growth in late spring. Boyce has pointed out (3, p. 6) that the fungus *Neofabraea malicorticis* (Cordley) Jackson, causing a biennial canker on apple trees in the Pacific Northwest, behaves in much the same way. Wilson (42) has published with considerable detail descriptions of the lesions caused by *P. pseudotsugae*.

With regard to the question concerning the origin of the Phomopsis disease of conifers, the authors are of the opinion that very probably *Phomopsis pseudotsugae* is a continental fungus, present in Europe in former years and occurring on a number of coniferous hosts either saprophytically or parasitically. It was collected by Rostrup in 1892 on *Abies pectinata* and has been recorded on this host and on European larch by Wilson (42); it also has been recently found as a parasite on *Cedrus atlantica* Manetti (1926), on *Larix sibirica* Ledeb. (1927) and on *Cedrus deodara* Loud. (1928). These trees are Old World species and it is very possible that one or more of these trees were the original hosts of the fungus. More than likely the organism was recognized in previous years but has been hidden under other identifications, e.g., *Phoma Pitya and P. abietona.*

Up to the present time the fungus is known to occur on the following hosts: the green form of Douglas fir, *Pseudotsuga douglasii*, the blue form *P. glauca*, the variety *caesia* of *P. douglasii, Larix leptolepsis* Murr. (Japanese larch), *L. europaea* DC. (European larch), *L. sibirica* (Siberian larch), *Abies pectinata* (silver fir), *Cedrus atlantica, C. deodara* and *Sequoia gigantea* Torr.[5] On *Cedrus atlantica* the fungus caused a definite girdling and a die-back of the terminal shoots. Among the hosts listed the greatest amount of damage occurs on the green Douglas fir. In Britain the blue form apparently is only occasionally affected. On the other hand, the latter host and particularly the intermediate forms of Douglas fir are repeatedly and seriously attacked by the American needle cast parasite, *Rhabdocline pseudotsugae* Syd., which, until 1927 (43), was confined to a single localized infection area in southern Scotland, but by that year was reported from several different sources in England (4). *R. pseudotsugae* is a very interesting case of an imported parasite which in its new environment generally attacks only one of the host forms which it ordinarily parasitizes in North America. Fortunately the more valuable green Douglas fir (Pacific coast form) has remained practically unaffected in Great Britain. The needle-cast disease is not known in Ireland or on the Continent.

[5] Specimens collected by Dr. E. P. Meinecke, Benmore, Argyllshire, Scotland, September, 1928. Culturally this form of *Phomopsis pseudotsugae* on Sequoia was identical with forms isolated from Douglas fir, larch, and cedar. Artificial inoculations made by Hahn with the *Cedrus deodara* form on nine-year-old potted trees of *C. atlantica* have given positive results.

In Norway, Hahn (1927) observed the blue form of Douglas fir vigorously attacked by *Phomopsis pseudotsugae,* which caused a serious die-back of the terminal shoots and branchlets extending throughout the tree. This injury was not accompanied by a typical trunk canker such as occurs on the green form of Douglas fir in Great Britain. A trunk and branch canker of common occurrence was observed however by him on the blue form associated with *Dasyscypha resinaria* (Cke. et Phill.) Rehm, and a *Dasyscypha sp.* (? *D. subtilissima* (Cke.) Sacc.). The parasitism of these Discomycetes has been briefly alluded to by Jørstad (17, p. 158–9) in his discussion of pine cankers. Further investigation of these attributed parasites appears to be necessary.

In consideration of the relationship between the parasite *Phomopsis pseudotsugae* and Douglas fir, the instance may very probably be considered one wherein an introduced host becomes subject to the parasitic attack of an organism already present in the country. Lesions of young and large trunks of the Douglas fir similar to those produced by *Phomopsis pseudotsugae* have not been observed hitherto in North America nor is there any record of the fungus occurring there. It appears unlikely that the fungus is an imported species introduced into Europe on Douglas fir from America.

Phomopsis pseudotsugae is now known to occur throughout Great Britain and Ireland. It was observed in Denmark by Boyce (1925), by Wilson (1926), and by Hahn (1927). Hahn (44) also collected the fungus in Norway and Sweden. In 1926 Wilson collected it in Holland. For practical purposes the Douglas fir parasite may also be considered as being present in France, Germany, and Belgium; for the presence of the organism in these countries is strongly suspected, although collections have not as yet been made and identified.

SUMMARY

An important disease causing canker and die-back of the Douglas fir has recently been described in Great Britain and attributed to *Phomopsis pseudotsugae.*

Certain investigators have regarded this fungus as synonymous with the older species of *Phoma pitya* and *P. abietina* which have been considered as being the cause of various diseases of the Douglas fir and other conifers. There also has been considerable confusion in mycological and forestry literature concerning the identity of these older species.

Critical investigation has shown that the three organisms are distinct. *Phoma pitya* is considered a species of Sclerophoma and has been named provisionally *Scl. magnusiana. Phoma abietina* is regarded as *Phomopsis abietina.*

Sclerophoma magnusiana is regarded as a parasite occurring on *Pinus sylvestris*. Typical *Phomopsis abietina* has been recorded only from Germany and France on Abies, where it is considered to be the cause of a girdling disease of small twigs and branches. *P. pseudotsugae* has been shown by preliminary inoculation experiments to be a true parasite.

Phomopsis pseudotsugae is regarded as an Old World species present on *Larix europaea, L. sibirica, Abies pectinata, Cedrus atlantica* and *C. deodara*. It was collected by Rostrup in 1892 on *Abies pectinata*, but identified at that time as *Phoma pitya*. It is now known to occur also on *Pseudotsuga douglasii, P. douglasii* var. *caesia, P. glauca, Larix leptolepsis*, and *Sequoia gigantea*.

The present known distribution of *Phomopsis pseudotsugae* includes Great Britain and Ireland, Holland, Denmark, Norway, and Sweden. For practical purposes, it must be considered to be also present in France, Germany, and Belgium. The Douglas fir parasite is not known to occur in North America.

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SEPTORIA ACICOLA AND THE BROWN-SPOT DISEASE OF PINE NEEDLES

GEORGE G. HEDGCOCK[1]

A serious needle blight of species of pine has been known in the southern United States for more than ten years and has been present at least since 1876. The increased interest in forestry in recent years has resulted in the discovery of its importance. It attacks most severely the needles of longleaf pine, *Pinus palustris* Mill., but is not confined to this species. The loblolly pine, *Pinus taeda* L., slash pine, *Pinus caribaea* More., and other species often suffer from it. The writer has made a study of this disease, but is unable to complete the work and will now publish the results to date for the use of others who may investigate it in the future.

This disease was first called by the writer the "red spot" needle disease in his notes and correspondence, and this name is used by Dearness (5) but it is now known by the more appropriate name "brown-spot needle disease" or "brown spot," because brown spots on pine needles are the most conspicuous symptom of its presence. It has also been called by others "rust" or "leaf rust" (2, p. 26–28) but these names, if used, will lead to confusion, since they already have been applied to the type of disease caused by forms of Peridermium belonging to the genus Coleosporium.

The first symptom of the disease is the appearance of a small, light gray-green, irregularly circular spot which changes quickly to a brown color and later encircles the needle in a narrow band, finally killing that portion beyond the point of attack. The dead portions assume a lighter brown color. In severe forms of the disease many brown spots form on each needle throughout its length, often killing it during the first season after infection. Each successive season's growth of needles is attacked, with the result that the trees are nearly defoliated every year and either die or are greatly retarded in their growth.

Chapman (2) reports greater losses from the "brown spot" in longleaf pine than from spring fires. He found 5.3 per cent of the seedlings up to six inches in height killed by the disease, 34.3 per cent of those from six inches to two feet, and 4.5 per cent of those two feet and over. He asserts that a defoliation of the trees for three successive seasons kills them. The writer has observed the same effect in many localities in Georgia, Florida, Alabama, Mississippi, Louisiana, and South Carolina. The disease is much more severe on trees in grassy, unburned tracts, a fact also noted by Chapman (2). After the trees are five feet or over in height, it causes only

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slight injury. Edgerton and Moreland (7) in 1923 reported a severe blight of longleaf-pine seedlings in Louisiana, which from their brief description is similar to and no doubt identical with the brown-spot disease.

ISOLATION OF CAUSAL ORGANISM

The pine-needle fungus, *Septoria acicola* (Thüm.) Saccardo, is constantly found fruiting on the older brown spots and on the older dead brown portions of the diseased needles. No other species has been found in constant association with the disease. Dilution cultures of *Septoria acicola* were made from pycnospores isolated from needles diseased with the brown spot and were grown on several kinds of agar media. Heavy, coal-black, stromatal areas were formed on the mycelium in cultures. Upon these stromata a crowded layer of coal-black subspherical pycnidia were found, which were typical of the genus Septoria. These pycnidia contained numerous smoky gray cylindric to curved spores, which at first were nonseptate but later developed from one to three septa, three septa being typical for the fungus. None of these spores were found to be viable, and all failed to germinate in poured plates. The fungus was propagated readily, however, by transferring bits of the mycelium from growing cultures to poured plates or slant tubes of agar media.

INOCULATION TESTS

Two sets of inoculations were made with *Septoria acicola* in the greenhouses at Washington, D. C. The first was made February 14, 1923, on the needles of healthy potted pine trees as follows: 4 *Pinus caribaea*, 2 *P. palustris*, and 2 *P. taeda* were inoculated in moist chambers of an iceless refrigerator (8) by laying on the trees in contact with the living needles, dead needles of *Pinus palustris* collected at Urania, Louisiana, January 27, which were badly diseased with the brown spot and heavily infected with *Septoria acicola* in fruiting condition. The trees were lightly sprayed with water and left for two days. Some of the needles of the 2 *P. caribaea* and 2 *P. palustris* bore ragged gray-green spots March 27, which browned lightly by June 2. On October 13 a few of the needles were dead or partly dead and of a light brown color. These bore fruiting bodies of *Septoria acicola*. Control trees remained healthy.

A second set of inoculations was made July 26, 1924, from spore-dilution cultures of *Septoria acicola* isolated from the needles of *Pinus palustris* diseased with brown spot, collected at Urania, Louisiana, January 7, 1924. Eighteen healthy potted trees of *P. palustris* were used; six trees were sprayed with the exuded spores of the fungus in a water suspension, six were sprayed with a maceration of the stromata and pycnidia in water suspension, and six control trees were sprayed with sterile water. All

were placed in the moist chambers of iceless refrigerators for two days. The six trees sprayed with the spore suspension and the six control trees remained healthy. Some of the needles of each of the six trees sprayed with the maceration suspension became diseased, showing brownish-gray spots in September, 1924, and by February 25, 1925, had either wholly or partly browned and died. The negative result with the spore suspension indicates that the spores were not viable. The positive result with the maceration suspension may indicate infection either from the mycelium or from the presence of viable spores remaining in the pycnidia. The brown spots produced were not quite typical of those produced in nature, but the blighted leaves were fairly typical and of a light brown color. The effect of the strong sunlight in natural infections probably tends to cause more intense browning than greenhouse light which enters through whitewashed glass and has an average intensity of about one-fifth of full sunlight. *Septoria acicola* was isolated from the diseased leaves from both experiments and grown in pure cultures from a spore dilution. The results of these experiments indicate that *Septoria acicola* is probably the cause of the brown-spot disease and that the fungus is probably parasitic.

TAXONOMY OF CAUSAL ORGANISM

De Thümen (13) described *Cryptosporium acicolum* Thüm. from a specimen collected by H. W. Ravenel at Aiken, South Carolina, in 1876 on *Pinus variabilis* Lamb. *P. variabilis* is a synonym of *P. echinata* Mill. There is a specimen in the Mycological Collections of the Department at Washington, D. C., collected by H. W. Ravenel (No. 1484, Mycotheca Universalis) on *Pinus variabilis* in 1876, which may or may not be a co-type. The pine, however, is not *P. echinata* but *P. caribaea* More. Saccardo (11, p. 507) in 1884 changed the name of *Cryptosporium acicolum* Thüm. to *Septoria acicola* (Thüm.) Sacc. on account of the septate spores. Geo. Martin (9) in 1887 reported the fungus on pine needles (Ravenel's collection ?) from South Carolina under the name *Septoria acericola.*

G. H. Martin (10) reported the brown-spot disease from Florida in 1922 on *Pinus taeda* and in 1923 from Louisiana on *Pinus palustris.* A published article in 1920 based on Hine's investigations (1), incorrectly attributed the brown-spot disease to *Lophodermium pinastri* (Schr.) Chev., which fruits on dead pine needles and occurs rarely on longleaf pine and only on dead fallen needles. *Lophodermium australe* Dearness is common but sparse on old gray needles of this pine clinging to the branches but is not associated constantly with the brown-spot disease. Chapman (2) in 1926 ascribed the disease to *Septoria pini* which is not known to occur on longleaf pine. A description of *Septoria acicola* follows:

Septoria acicola (Thüm.) Sacc. Syl. Fung. 3: 507, 1884.

Syn.; *Cryptosporium acicolum* Thüm. Fungi Am. Trig. sp. nov. Flora 61: 178, 1878 (13).

Actinothyrium marginatum Sacc. Nuov. Giorn. Bot. Ital. 27: 83, 1920 (12).

Lecanosticta pini Syd. Ann. Myc. 20: 211, 1922 (12).

Lecanosticta decipiens Petr., Ann. Myc. 20: 214, 1922 (12).

Lecanosticta acicola (Thüm.) Syd. Ann. Myc. 22: 400, 1924 (12).

Acervuli amphigenous, globose at first, 50–100 μ in diameter, becoming elongate up to 1.5 mm., seated in the mesophyll, discharging spores through a cleft in the epidermis. Basal layer of colored compact pseudoparenchyma.

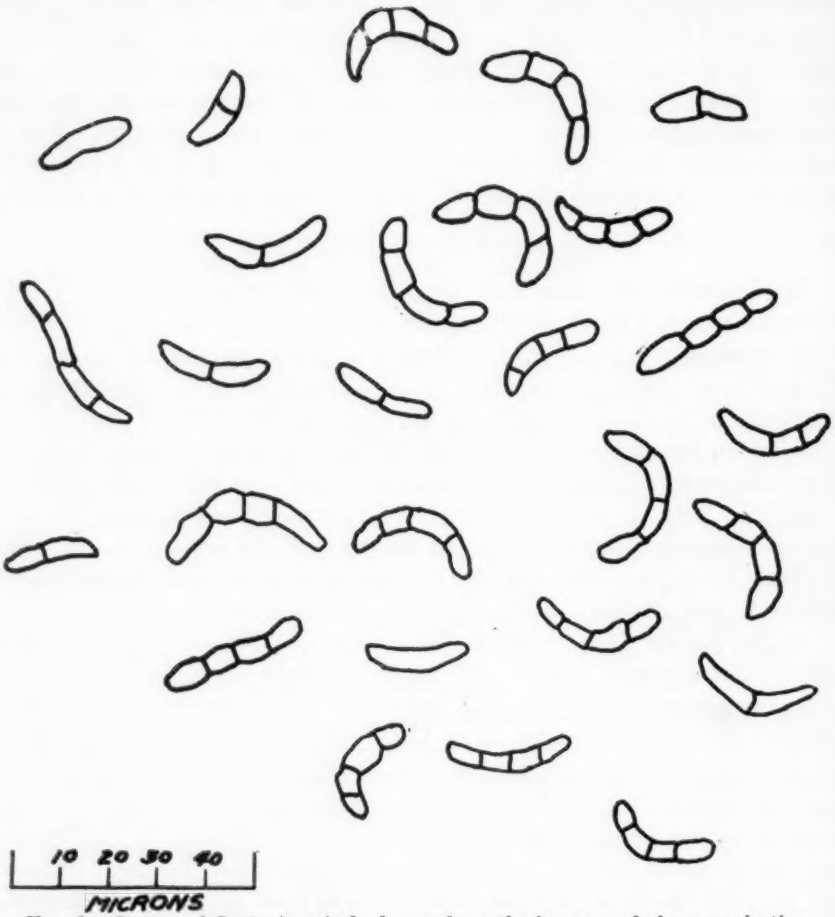

FIG. 1. Spores of *Septoria acicola* drawn from the image made by a projection apparatus.

Spores simple, subhyaline, becoming 1–3 septate and smoky, often curved more in one half than the other, mean of 100 spores 29.5 by 3.8 μ with a sextile range[2] of 32–27 by 4–.5 μ (Fig. 1). The acervuli occur on brown spots forming on living pine needles and also on other lighter brown portions of dead needles.

On the needles of *Pinus palustris*, F. P. No. 41664, collected by Freda Detmers at Bogalusa, Louisiana, January 7, 1924.

Sydow and Petrak (12) have some basis for putting *Septoria acicola* in the genus Lecanosticta, since it differs in its natural form of fruiting from typical species of both Cryptosporium and Septoria. In artificial cultures, however, it resembles closely species of Septoria and the writer chooses at present to leave it in the genus Septoria.

Dearness (4) has found an ascus-bearing species, *Oligostroma acicola* Dearn., closely associated with *Septoria acicola* in a number of southern collections made by the writer and considers it to be probably a perfect stage of the latter fungus. Cultural proof, now lacking, will be necessary to establish this relationship.

HOST RANGE AND GEOGRAPHICAL DISTRIBUTION

In the collections of the Office of Forest Pathology at Washington, D. C., there are 121 collections of *Septoria acicola* and the brown-spot disease on ten species of pine from 11 States, and in the Mycological Collections of the Department there are 11 more collections with two additional host species. The number of collections on each species of pine for each State is now given.

On *Pinus attenuata* Lemm., Ore. 1.

On *P. caribaea*, Fla. 4, Miss. 1, and S. Car. 1.

On *P. echinata* Mill., N. Car. 2, and S. Car. 1.

On *P. glabra* Walt., Fla. 3.

On *P. nigra austriaca* Schneid., Kan. 1, Ky. 1, and Mo. 1.

On *P. palustris*, Ala. 2, Fla. 41, Ga. 8, La. 13, Miss. 5, N. Car. 2, S. Car. 9, and Tex. 1.

On *P. ponderosa* Laws., Idaho 3.

On *P. rigida* Mill., N. Car. 2, Pa. 1, Tenn. 1, and Va. 1.

On *P. serotina* Michx., S. Car. 2.

On *P. sondereggii* Chapm. (*P. palustris* × *P. taeda*), La. 1.

On *P. taeda*, Ark. 1, Fla. 5, Ga. 1, La. 2, Miss. 1, N. Car. 1, S. Car. 6, Tenn. 3, and Tex. 1.

On *P. virginiana* Mill., Ga. 1, and N. Car. 2.

From the data given, it will be seen that the present known range of *Septoria acicola* and of the associated brown-spot disease in the eastern United

[2] The "sextile range" is that remaining after the sixth of the spores that are smallest and the sixth that are largest have been discarded.

States is from Pennsylvania west to Kansas and south to Florida and Texas, and in Oregon and Idaho in the northwest.

CONTROL OF THE DISEASE

Fire destroys the spores of *Septoria acicola* and must to some extent hinder the spread of the disease; at the same time, however, it kills many of the small pine trees. The effect of the fungus versus the effect of fire is still a subject of controversy among foresters. It is not advisable to recommend the burning of the ground cover of areas with diseased pines for the purpose of destroying the spores of the fungus until the facts are more fully known. The subject of the effect of fire in controlling the disease has been discussed in a number of articles by Chapman (2, 3), Hine (1), Demmon and Hadley (6), and Wyman (14). A few data from experiments have been published (1, 2, 3, 14) but the results are too limited in extent to form a basis for a final conclusion. Experiments from a number of localities widely separated and under varying conditions will be necessary to finally ascertain the correct method of handling diseased areas.

The use of fungicides for the control of the brown-spot disease in forest-tree nurseries is advised. The following method, suggested by the writer, was used in young stands at Bogalusa, Louisiana, in the spring of 1928. Spray every two weeks with a spraying outfit that gives a misty spray, beginning when the young needles emerge and ceasing when the leaves have attained their growth. Use standard Bordeaux 4–4–50 with the addition of two pounds of either whale-oil soap or a casein soap like "Kayso" to every 50 gallons of spray solution. Ordinary Bordeaux without the soap rolls off pine needles without adhering to them. The use of Bordeaux and of lime-sulphur sprays in this manner at Bogalusa controlled the disease as long as the spraying was continued. The period suggested by the writer was too short. Spraying should continue longer.

Foresters, plant pathologists, and others interested in the brown-spot disease are requested to send specimens to Paul V. Siggers, Associate Pathologist, Southern Forest Experiment Station, New Orleans, La. Mr. Siggers will continue the study of this disease.

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THE RELATION OF LIME-SULPHUR SPRAYS TO THE ABSCISSION OF YOUNG APPLES

F. S. Howlett and Curtis May

Strong lime-sulphur solutions, sprayed on young apple foliage and fruits, may cause marginal burning of the leaves, cupping of the blades, curvature of the midrib, burning out of scab spots, and deformity of the young apples. This type of injury has been recognized for a long time. Recently, it has been stated that excessive abscission of young apple fruits may be caused by lime-sulphur spray, even though there is no visible spray injury on the leaves or fruit.

Bagenal and others in England (1) concluded from their spray experiments in 1924 that Bordeaux mixture 4–4–50 did not increase the fruit drop from a healthy tree, but that lime-sulphur 1–29 induced an excessive amount of it. They state that there was no serious spray injury of leaves or fruit visible to the unaided eye. They add that "the unusual percentage of drops due to spraying with these two fungicides cannot be exactly determined from evidence available, owing to the variation shown by the control trees."

Dutton's (2) experiments were largely concerned with the effect of lime-sulphur and Bordeaux sprays on the set of fruit when some leaf fall occurred. He stated that the June drop was excessive for trees sprayed very heavily with lime sulphur but was normal for the Bordeaux-sprayed trees.

The present study was designed to determine the effect of the second after-bloom spray on abscission of young fruits when there were no visible marks of spray injury either on them or the foliage.

Method

The trees used were healthy, moderately pruned, 27-year-old Ensee and Grimes Golden varieties growing in the grass-mulch orchard of the Ohio Agricultural Experiment Station at Wooster. Each was given an annual pre-bloom application of 5 pounds of nitrate of soda. These varieties were selected, not only because they are susceptible to spray injury, but also because they bear flowers on shoots growing sufficiently long to be easily bagged or individually sprayed.

Three pre-blossom sprays and a calyx spray were applied in 1927. The following season the trees were dusted up to and including the calyx application.

Liquid lime sulphur at dilutions of 1 to 15, 1 to 40, and 1 to 60 was tested the first season. The following year liquid lime sulphur, 1 to 60, 1 to 100, and 1 to 120, and Bordeaux mixture, 2–4–50 and 1–2–50, were used.

After the fruits had passed through the first drop the number of fruits in each cluster was reduced to two laterals (Fig. 1). Those left on the clus-

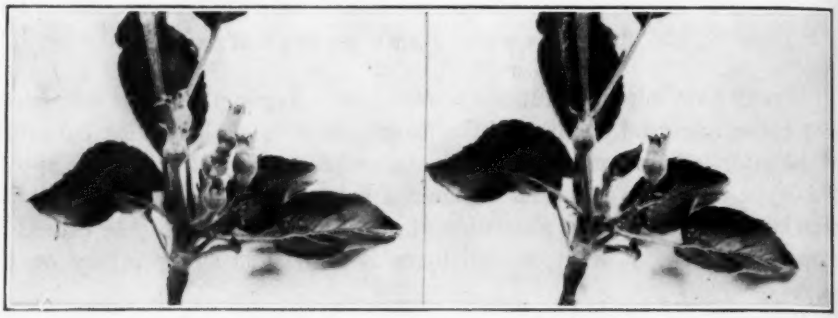

FIG. 1. Left, cluster of Ensee before thinning, 1928. Right, same cluster after thinning to 2 laterals.

ters showed every evidence of having successfully passed the first wave of dropping. The elimination was done each year by one person and over the various limbs an attempt was made to alternate the clusters receiving the various treatments.

In 1927, the clusters were sprayed individually by means of a hand-barrel pump. A galvanized-iron pan was made with a hole in the center of the bottom. This hole could be closed by a crescent-shaped slide. There was also a reservoir underneath to catch the drip. Before spraying the cluster was placed through the opening of the pan, after which the hole was almost completely closed by means of the slide. The cluster was then well drenched with spray. The drip was retained by the reservoir.

In 1928, approximately 750 fruit clusters were selected on each tree and the fruits reduced, as before, to two per cluster. These clusters were divided into three sets and the sets marked by labels of different colors. Two sets of clusters were covered with glassine bags. The tree was then sprayed with the regular orchard sprayer (375 lbs. pressure) using the stronger dilution of lime sulphur. As soon as the selected clusters of Set 1 had dried they were bagged. The bags were then removed from Set 2, and the tree again sprayed with a weaker solution. The bags were then removed from the clusters of Sets 1 and 3. Two spray dilutions and an unsprayed check were obtained on each tree by following this procedure. In both years the elimination, bagging, and experimental spraying were completed within 24 hours. The data were taken shortly after the June drop was completed.

RESULTS

The results of the tests of 1927 are given in table 1. It is to be observed that the percentage of fruit set of all the sprayed clusters of both trees was

TABLE 1.—*The effect of lime sulphur on abscission of young Ensee apples. Wooster, 1927*

Tree	Dilution of spray	No. of clusters	Percentage set of fruits	Percentage of clusters with		
				0 fruit	1 fruit	2 fruits
411–3	1–15	93	51.6	22.6	51.6	25.8
	1–40	96	60.4	16.7	45.8	37.5
	1–60	89	60.7	16.8	43.8	39.4
	unsprayed	102	84.8	4.9	20.6	74.5
411–2	1–15	82	61.0	17.1	43.9	39.0
	1–40	94	68.1	10.6	42.6	46.8
	1–60	100	67.0	16.0	34.0	50.0
	unsprayed	99	93.4	0.0	13.1	86.9

lower than that of the unsprayed clusters. In order to obtain the odds that the differences found were not due to chance the variability of the determinations was measured by dividing the samples at random into sub-samples of approximately 50 clusters each. The deviations of these sub-samples from their respective sample means were squared. From these values t, as given in Fisher (3), was computed for the various differences. The values of t were converted into odds by the use of Student's tables (5).

Combining the data of the two trees the odds that the differences obtained in 1927 between the clusters sprayed with 1 to 60 solution and the unsprayed

TABLE 2.—*Effect on fruit abscission resulting from spraying young apples with lime-sulphur or Bordeaux mixture. Wooster, 1928*

Apple variety	Fungicide	No. of clusters	Percentage set of fruits	Percentage of clusters with		
				0 fruit	1 fruit	2 fruits
411–1	Lime sulphur 1– 60	222	73.4	5.9	41.4	52.7
Ensee	" " 1–100	223	76.9	5.4	35.4	59.2
	Check	215	83.3	3.6	26.1	70.3
409–4	Lime sulphur 1– 60	174	31.9	48.8	38.5	12.6
Grimes	" " 1–100	187	51.3	20.9	55.6	23.5
	Check	216	48.1	27.8	48.1	24.1
409–5	Lime sulphur 1– 60	172	59.4	17.1	46.8	36.1
Grimes	" " 1–120	169	63.7	12.8	47.1	40.1
	Check	195	69.0	10.3	41.5	48.2
409–7	Bordeaux 2–4–50	222	55.2	16.8	56.2	27.0
Grimes	" 1–2–50	222	46.8	26.7	51.1	21.1
	Check	224	58.2	22.3	48.2	29.5

clusters were not due to chance are 332 : 1.[1] There were no significant differences between the clusters sprayed with the 1 to 40 and the 1 to 60 dilutions.

The data for 1928 are given in table 2. Ensee tree 411–1 is a companion tree of those used in 1927. The data indicate that the 1–to–60 (odds 1000 : 1) and the 1–to–100 (odds 155 : 1) dilutions caused slight fruit abscission on this tree.

On Grimes Golden tree 409–4 there was a marked reduction (odds 908 : 1) from the clusters sprayed with 1 to 60, but none from those sprayed with 1–to–100 lime sulphur. On tree 409–5 the odds that the set reduction caused by 1–to–60 was significant were 9.05 : 1. The 1–to–120 dilution did not cause a significant loss.

If the data from these three trees are combined and the odds calculated, the results are as follows:

1–to–100 and 1–to–120 vs. unsprayed	4.8 : 1
1–to– 60 vs. "	832 : 1
1–to–60 vs. 1–to–100 and 1–to–120	184 : 1

It is apparent that 1–to–60 lime sulphur reduced and that 1–to–100 or greater did not reduce the set of fruit on these trees. However, there was a significant difference between the 1–to–60 and the 1–to–100 dilutions. Since the former caused fruit abscission and the latter did not, we may conclude that the dilution at which there would cease to be significant injury would be between these two dilutions.

The loss of fruit due to 1–to–40, 1–to–60, or greater dilutions of lime sulphur was not great enough to affect their normally heavy crop. If trees of these two varieties are of the usual commercial vigor they have a very heavy set, even after the June drop is completed, and thus require considerable thinning.

The results from the tree sprayed with Bordeaux were inconclusive. Because of the heavy rains subsequent to the application of the spray, there were a considerable drip of Bordeaux and a consequent russeting and deformity of many of the fruits of the check clusters.

The great extent to which the second, or June, drop was reduced in Ensee on the unsprayed clusters by reducing the fruits to two is indicated by the relatively high percentage sets (Table 1) of the check clusters, 84.8 and 93.4 per cent, respectively. The fruit drop was not so greatly reduced on Grimes Golden as on Ensee. However, unpublished data of the senior writer indicate that the percentage of fruit retained by the treated clusters was greater than that of clusters whose fruits were not reduced before the June drop.

[1] The writers wish to thank Mr. Marion T. Meyers for his assistance in the statistical treatment of the data.

DISCUSSION

There is a well-known correlation between lime-sulphur and Bordeaux spray injuries and the weather conditions at the time of and immediately following the application of the spray. The maximum and minimum temperatures for the two weeks following the application of the experimental sprays are given in table 3.

TABLE 3.—*Maximum and minimum temperatures (Centigrade) for 2 weeks following the application of the experimental sprays. Wooster, 1927, 1928*

	May, 1927						June, 1927							
	26	27	28	29	30	31	1	2	3	4	5	6	7	8
Max.	22.7	16.1	15.0	18.8	20.0	27.7	21.1	16.1	17.7	20.0	25.0	16.1	17.7	20.0
Min.	12.7	6.6	3.8	3.8	10.0	3.8	6.1	3.8	2.7	8.8	17.2	6.6	7.7	5.0

	June, 1928													
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Max.	21.1	22.7	18.3	20.0	22.7	20.5	19.4	18.8	10.0	20.5	17.2	20.0	25.5	28.8
Minn.	4.4	8.3	9.4	2.7	11.1	12.2	10.0	10.0	17.7	11.1	7.2	5.5	10.0	20.5

On none of the days on which sprays were applied was the temperature high enough to cause visible fruit or foliage injury when the standard strength of lime sulphur was used. It is generally regarded that 26.5° C is the danger point for lime-sulphur-spray burn. In 1927 it was five days, and in 1928 two weeks after the spray was applied, before this temperature was recorded.

In general, the weather during the periods following the spraying in both years was cool. Cool weather is supposedly not conducive to lime-sulphur injury. Bordeaux is, however, particularly injurious in cool, wet weather. Such a period followed the spray applications of 1928 and severe russeting and dwarfing of the fruit occurred on the Bordeaux-sprayed trees.

The data given by Bagenal (1) and Dutton (2) indicate that they consider the second after-bloom spray to be the one responsible for excessive abscission of apple fruits. This, the writers believe, is due to the fact that the spray is applied usually, if not always, just previous to or during the June drop of the varieties employed in the experiment. The data in table 4 indicate that this is the case at Wooster.

Each year normal dropping continued for two to three weeks after the application of this spray. The writers wish to point out that mere observa-

TABLE 4.—*Relation of the time of after-bloom sprays to end of fruit abscission. Wooster, 1924–1928, inclusive*

Year	Dates of application		Date of period of June drop
	Petal-fall spray	Two-weeks spray	
1924	May 22–29	June 9–13	June 6–July 7
1925	May 8–16	May 26–June 4	June 4–July 1
1926	May 26–31	June 10–17	June 10–July 7
1927	May 12–17	May 31–June 2	June 2–July 1
1928	May 25–29	June 8–13	June 9–July 1

tion of fruits falling during or after either the calyx or 2-weeks' spray is not, in itself, evidence that the abscission is due to spray injury.

The relation of varietal differences in fruit-setting characteristics to food competition among the fruits of a cluster deserves consideration in a study of the relation between spray injury and abscission (4). Immediately after petal fall on Ensee and Grimes Golden 3 to 5 flowers in a cluster usually enlarge and survive the first drop (figure 1). The enlargement is marked usually about a week before the normal time of application of the second after-bloom spray (2–3-weeks' spray). Varieties like Ensee and Grimes Golden which pass through the first drop with a heavy set commonly have a heavy June drop. This drop appears to be due largely to food competition and unless some means are taken to control this factor, it would appear to be very difficult to obtain reliable data on this subject. The extent of the reduction of the June drop by reducing the fruits to 2 per cluster has already been pointed out. It is highly probable that the differences between the set of fruit on the sprayed and unsprayed clusters were due to the materials used and not to loss of fruit because of insufficient food supply.

Trees of the same age and apparently the same vigor may vary in their ability to hold their fruit. This is shown by the data (Table 2) for the unsprayed clusters of Grimes Golden trees 409–4 and 409–7. Both trees appeared to be of equal vigor but they had June drops of different degrees of severity. Spraying one tree and not another and comparing the drops from each, and assuming that differences obtained are due to the spray treatment, are open to criticism. Tree variation is such an important factor that the spraying of a few trees and comparing them with unsprayed trees are rather unsatisfactory procedures.

The writers make no attempt to apply the results of this study to any but the varieties used. Ensee and Grimes Golden are susceptible to spray russeting of the fruits and possibly are more affected by lime-sulphur solutions than other heavy-setting varieties.

SUMMARY

1. Lime-sulphur and Bordeaux mixture were applied as second after-bloom sprays to determine whether they caused excessive fruit abscission when there was no visible foliage injury.

2. The number of fruits in the individual clusters was reduced to two to eliminate food competition as a factor in fruit abscission.

3. The different spray dilutions were applied to the same tree to eliminate individual tree variation.

4. Lime-sulphur solutions of 1–40 and 1–60 dilutions caused some abscission of fruit from Ensee and Grimes Golden trees.

5. Lime-sulphur solutions of 1 to 100 or greater dilution did not cause significant abscission of fruit from Ensee and Grimes Golden trees.

6. The abscission due to any of the spray dilutions was not sufficiently great to reduce their normally heavy commercial crops.

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IS THE ASTER-YELLOWS VIRUS DETECTABLE IN ITS INSECT VECTOR?

IRENE D. DOBROSCKY

The leaf hopper, *Cicadula sexnotata* Fall., has been shown to be the specific carrier of the virus of aster yellows (6). Furthermore, this virus must remain in the body of the insect for at least ten days before it can reinfect another plant. Similar conditions have been found to obtain in the case of curly top of sugar beets and the leaf hopper, *Eutettix tenellus* (Baker) (8); and, also, in the case of streak of maize and the leaf hopper, *Balclutha mbila* Naude (9). The necessity for an incubation period of the virus would seem to indicate that we are dealing with an organism which must either multiply in the insect or undergo a definite part of its life cycle there. This simulates closely the facts known about malaria, yellow fever, Texas cattle fever, nagana disease of cattle, and African sleeping sickness.

Because of the long incubation period of the aster-yellows virus in its insect vector it was thought this leaf hopper might be a favorable one in which to detect the virus. An intensive study of the morphology and cytology of the insect was therefore undertaken. Such a study might result in the discovery of the causative agent of yellows or some evident reaction of the insect to the virus. It might result even in the discovery of an intracellular body associated with the virus in the insect.

Botanists who have studied the tissues of plants affected with virus diseases have found intracellular bodies in connection with many of these diseases. At least twenty-eight species of plants having the mosaic type of virus disease have been reported as containing these so-called "x-bodies," or inclusion bodies.

Plants affected with the yellows type of virus diseases, such as aster yellows, peach yellows, curly top of sugar beets, and cranberry false blossom, have not been found to contain intracellular bodies. The morphological symptoms of a plant affected with aster yellows are, however, very striking and characteristic. Nevertheless, no bacterium, protozoan, or fungus has been found associated with the disease. The causative agent has not been found in those plants susceptible to the aster-yellows virus. It seemed advisable to search for it in the insect.

METHODS

The histology and cytology of the leaf hopper, *Cicadula sexnotata*, were studied by making smears and examining these under dark-field illumination, by gross dissections of the alimentary tract, and by serial paraffin sections.

Smears. In making smears about fifty adults, hatched and reared on diseased asters, were used. Similar virus-free adults served as checks. Each insect was slowly crushed between a slide and a cover slip and, while the smear was still wet, it was examined under dark-field illumination. Numerous globules of fat were observed, some showing Brownian movements. There was no indication of vital movement shown by any of the bodies in the smears.

When the smears had dried they were stained with Wright's blood stain for one minute. All the slides showed small brownish granules which were the pigment granules of the compound eyes. About one-half of the smears contained oval-shaped bodies 8 μ to 12 μ long, with pink capsule-like rims, blue cytoplasm, and bright red nuclei. A few forms were found dividing. These bodies resemble the spores of Cladosporium, a soil-infesting fungus. As the spores were never found in the paraffin sections, it is probable that the insects bore them externally. Nuclei of all sizes and stages of activity were found in the smears. Crushed nuclei in the spireme stage took a pink stain and resembled clumps of bacteria. Many fragments of broken sperms were found but there was nothing present resembling either a bacterium or a flagellate.

Dissections. Gross dissections were made to study the alimentary canal. The stomach was found distended with a dark brown fluid. Continuing from the stomach is a narrower portion of the midintestine. This is very white and opaque in appearance, due to the accumulation of calcium-carbonate crystals within the cells. It is thought that the liquid food passes by osmosis from the stomach to the posterior intestine without passing through this narrow portion of the midintestine. Two of the most important appendages of the alimentary canal are the salivary glands and Malpighian tubules. The Malpighian tubules are four in number, rather large and thick. The salivary glands are in the head and prothorax. They are so small and so transparent that it is difficult to find them.

No cysts, lesions, or abnormal enlargements of any organ were found in the course of this macrodissection.

Paraffin Sections. More than a thousand insects, adults, and nymphs were fixed in various reagents, including Gilson's, Regand's, Bouin's, Flemming's weak, and Carnoy's solution. The sections were cut at 4 μ or 7.5 μ. They were then stained with Wright's, Giemsa's, Wolbach's Modified Giemsa, Heidenhain's Haemotoxylin, Flemming's, Mallory's Methylene blue, Pianese III B, Gentian violet, and Safranin, or acid fuchsin stain. For secretory cells of the salivary-gland type, Gilson's fixative followed by Wright's Romanowsky stain gave the best results. The Romanowsky stain, as put up by Coleman and Bell Co., proved most satisfactory. McNeal (7) has recently shown that there are four essential dyes in this stain. They are eosin, methylene blue, methylene azure, and methylene violet.

OBSERVATIONS

The parasites which cause malaria are found in cysts in the stomach walls and also as free swimming flagellates in the salivary glands of their mosquito hosts. Herpetomonas associated with oriental sore was found in the salivary glands of the fly *Phlebotomus papatasi* (1). Holmes (5) found *Herpetomonas elmassiani* (Migone) in the salivary glands of *Oncopeltus fasciatus* Dall., a bug which feeds on milkweeds. The flagellate, *Leptomonas davidii* Lafont, was found by França (4) in the salivary glands of the hemipteron *Stenocephalus agilis* Scop. which feeds on Euphorbias. These organisms are all ingested by their insect hosts. In several insect-borne diseases the causative organism has been found in the alimentary tract, proper. It seemed advisable, therefore, to make an intensive study of the salivary glands and the alimentary tract of the leaf hopper in question in the hope of finding some indication of the presence of the virus.

Salivary glands.[1] The salivary glands lie beneath the brain and occupy the dorsal floor of the head. They are made up of equal halves lying on either side of the œsophagus. Each half resembles a small bunch of grapes with a fore-shortened central stem. The lobules, twenty-three in each half, are large secreting cells containing two nuclei. Physiologically, the salivary glands may be divided into three types of cells, the mucous cells, the serous cells, and the cells which line the accessory or reservoir glands.

In making a comparative study of the salivary glands of healthy and viruliferous individuals, the following characteristics were noted: 1. Size, shape and staining properties of each lobule; 2. Texture and staining reaction of cytoplasm; 3. Vacuoles and inclusions in cytoplasm; 4. Size, staining properties and physiological state of nuclei; 5. Amount of secretion.

Though several hundred insects were examined with these points in mind, no constant difference was observed. No lesions, no cysts, no difference in size or shape of lobules, nor any alteration in the physiology of the cells, as indicated by their staining reactions, could be found.

After this study of the cytology of the secreting cells, the author proceeded to look for any foreign organism that might be present. The numerous stains used were calculated to detect the presence of bacteria, fungi, protozoa, Rickettsia or x-bodies. None of these was found in the salivary glands of either the normal or the viruliferous individuals.

In one experiment a series of adults, fed from one to twenty-three days on diseased asters, was studied in the hope of finding some progressive symptoms to account for the incubation period of the virus. No symptom indicative of the presence of the virus could be found.

[1] A more detailed morphological description of the salivary glands will be published by the author in another paper.

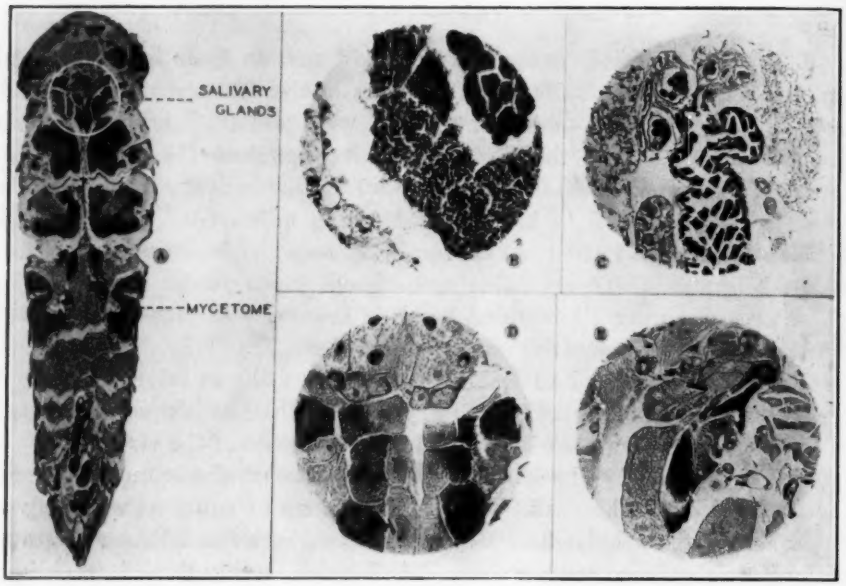

FIG. 1. A. Longitudinal section of adult female of *Cicadula sexnotata*. Shows salivary glands in prothorax and mycetome on either side of the first and second abdominal segments. (7 μ thick, × 50.)

B. Mycetome in detail. Note zone of empty cells with small marginal nuclei. (7.5 μ, thick × 430.)

C. Midintestine of disease-carrying insect. (7 μ thick, × 180.)

D. Salivary glands of adult insect starved ten hours. (7 μ thick, × 200.)

E. Lateral view of salivary glands of healthy normal insect. (7 μ thick, × 160.)

In another experiment some adults were starved and their salivary glands compared with those of normal insects in order to see how the cells were altered. It was found that the individual cells of the glands of starved insects were larger, due to an accumulation of secretion. This is illustrated in figure 1, D. Figure 1, E, shows the salivary glands of a normal healthy insect.

Alimentary tract. It is a well known fact that most insects shed their chitinous covering several times during the course of their development. The anterior and posterior intestines are lined with a thin intima, a continuation of the outer heavy chitin. This intima is shed along with the outer coat when the insect molts. An insect which has fed on a diseased plant and becomes infective does not lose its infective principle in spite of this process of shedding. Since the midintestine is not lined with intima, it would seem that this is the logical place to look for obligate parasites. Figure 1, C, is a photograph of the anterior portion of the midintestine, showing the large darkly stained cells which compose it.

It has been found in the case of several insect-borne diseases that the parasites are first localized in some part of the alimentary canal and from there get into the salivary glands. *Cicadula sexnotata* was found remarkably free from all parasites. Its alimentary tract does not harbor any foreign organisms.

Cowdry (3) examined one hundred and eleven species of insects and found that nineteen contained Rickettsia bodies. The writer, using Cowdry's methods of staining and fixing was unable to find any Rickettsia in the body of this leafhopper.[2]

Mycetome. The mycetome is an organ present in most Homoptera. This might easily be mistaken for a sign of the disease in the insect under discussion. Witlazil (11) regarded it as excretory in function, others ascribed to it a nutritive function. Sulç (10), Buchner (2), and Glaser, however, have concluded that the organ is parasitic.

The mycetome is a large paired organ located in the first two abdominal segments, as shown in figure 1, A. The cells composing it are closely packed with organisms of a fungal nature, whence the organ derives its name.

Three distinct zones, comprising different types of cells, can be seen in this organ. Figure 1, B, an enlargement of a portion of the mycetome, shows these zones very clearly. In the center of the organ are numerous compact cuboidal cells. The nuclei are almost one-half as large as the cell, and the cytoplasm is densely filled with a mycelium-like growth. With Wright's stain this zone is colored dark blue. The next zone is composed of very much larger cells with smaller nuclei. The cytoplasm is obscured by the presence of the thick mycelium-like strands. This zone stains a pinkish purple. The third zone is one-cell thick, the cells being almost empty of cytoplasm. Nothing but small, compact, dark blue nuclei are to be found along the borders of these outer cells.

The whole organ is embedded in the fat tissue and has no direct connection with any other organ. It is present in virus-free as well as virus-bearing individuals. The organisms within this mycetome are supposed to migrate to the eggs and are so transmitted from one generation to another. Since this organ is hereditary and since it is not in communication with any other organ, it is not likely that it can be concerned in the problem of the insect's ability to harbor the yellows virus.

DISCUSSION

The virus diseases of plants are essentially insect-borne. Sucking insects, especially aphids and leaf hoppers, are the chief disseminating agents

[2] Dr. Cowdry was kind enough to examine some of the writer's slides and confirmed this conclusion.

under natural conditions. Of the leaf hoppers known to be virus vectors, all but one are very closely related in the classification of insects.

Up to the present time, no one has done any intensive research on the morphology and cytology of these insect vectors with the view to locating the virus. The writer has been unable to find any histological or cytological differences between virus-free and virus-bearing individuals of the species *Cicadula sexnotata*. While the tecnique used has given only negative data, it is possible that other methods of fixing and staining may yield positive results.

A study of the other insect carriers of plant viruses may give a clue as to where the yellows virus may be located in the body of the insect. Physiological studies may also throw light on the subject. Significant results might be obtained by making determinations of the pH of individual insects. If certain portions of virus-carrying individuals were isolated and healthy insects made to feed on them, one might get an indication of the whereabouts of the virus. Several attempts have been made to culture the virus in plant decoctions. Some experiments in culturing the virus in insect juices should also be undertaken.

SUMMARY

A study of smears of virus-bearing insects under dark field illumination, of gross dissections, and of paraffin sections did not reveal any visual evidence of the presence of the aster-yellows virus in *Cicadula sexnotata*.

The mycetome, a hereditary parasitic organ, is evidently not concerned in the problem of the virus-carrying ability of the insect.

After an intensive study of the salivary glands and alimentary tract of *Cicadula sexnotata* with a view to finding the causative agent of aster yellows, the writer was unable to find such an agent or any lesions which might be due to the presence of such an agent. No bacterium, Rickettsia, fungus, protozoan, or x-body was found which could be considered of etiological significance.

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LIFE HISTORY AND PATHOLOGICAL ASPECTS OF GODRONIA CASSANDRAE PECK (FUSICOCCUM PUTREFACIENS, SHEAR) ON CRANBERRY

C. L. Shear and Henry F. Bain

End rot is now known as the most important fruit rot of cranberries. The pycnidial form of the fungus causing end rot was described by the senior writer in 1917 as *Fusicoccum putrefaciens* (5), and it was suggested that a Discomycete, occasionally associated with the pycnidia about the blossom end of mummied berries, was perhaps the ascogenous stage of the fungus. The earlier studies of the end-rot organism were confined to the eastern United States, where ascocarps of the Discomycete were but rarely found and viable ascospores were difficult to obtain.

B. A. Rudolph succeeded in producing typical pycnidia of Fusicoccum from cultures made from the tissues of an immature apothecium of this Discomycete found on a decayed berry in Massachusetts in 1917. Three years later the senior writer was able, through the assistance of Rudolph, to obtain some fresh ascocarps from the Pacific Coast, but was unable to obtain single-spore cultures. Cultures made by transplanting asci and paraphyses after careful surface sterilization all showed the lemon-yellow to orange-citrine color characteristic of Fusicoccum as illustrated in the colored plate accompanying the first description (5), and several cultures finally produced typical pycnidia of the fungus.

While carrying on cranberry-disease investigations in Oregon and Washington in 1923, the junior writer found an abundance of mature apothecia with viable ascospores. Twenty single-ascospore cultures were made, partly from apothecia on berries and partly on stems. Germinating ascospores are shown in figure 6, B. Germ tubes most frequently arise from the terminal cells of the spores, although it is not uncommon for one or more intermediate cells to produce germ tubes simultaneously. These cultures all showed the typical mycelial color and general appearance and finally produced typical pycnidia of *Fusicoccum putrefaciens*. No apothecia, however, have ever developed in any of these or other single-ascospore or pycnospore cultures. The evidence already given, however, seems sufficient to prove the genetic relation between the two forms of the fungus.

After many attempts to identify the apothecial form of the organism by comparison with various Discomycetes which have been found on Vaccinium and closely related Ericaceous plants, it was finally found to agree with *Godronia cassandrae* Peck (2, p. 50), which was described from specimens collected on dead branches of *Cassandra calyculata* L. at Karner, N. Y., in August, 1885. The senior writer recently found excellent specimens of this

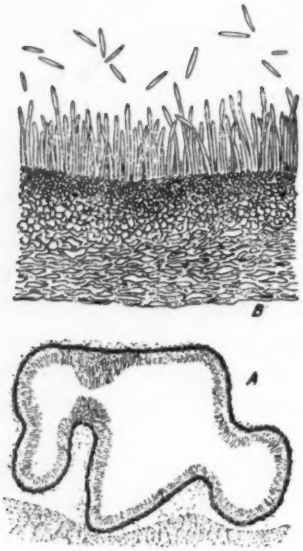

FIG. 1. *Fusicoccum putrefaciens*: A, Vertical section of a chambered pycnidium from a corn-meal agar culture, X 43.5; B. part of section of pycnidium grown in pure culture on sterile sweet clover stem (*Melilotus alba*), showing sporophores and spores, X 420. After Shear.

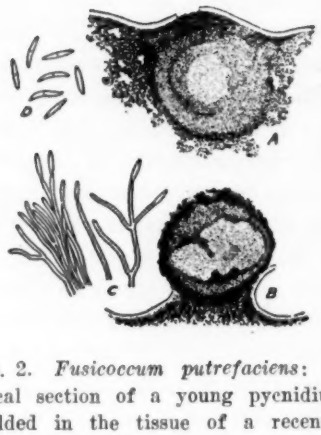

FIG. 2. *Fusicoccum putrefaciens*: A, Vertical section of a young pycnidium embedded in the tissue of a recently rotted cranberry; B, vertical section of a nearly mature pycnidium showing two chambers, X 43.5; C, separate branched and simple sporophores with immature spores; D, separate spores, showing variations in form and size, X 420. After Shear.

fungus in both stages on Cassandra at Upper Saranac and Schoon Lake, N. Y., and Lakehurst, N. J. This material agrees with that found on both the fruit and dead stems of the cranberry and also with Peck's original description of *Godronia cassandrae* which is, for convenience, quoted in full below:

GODRONIA CASSANDRAE N. SP. PECK

Receptacle small, .02 to .03 inches [.5 to .7 mm.] broad, sessile or nearly so, depressed, urceolate, tawny brown, the hymenium whitish or livid when moist, darker when dry, the narrow mouth entire or slightly dentate-lacerate, almost closed when dry; asci cylindical, .004 to .005 inches [101 to 127 microns] long, .0003 to .0004 inches [7.6 to 10.1 microns] broad; spores filiform, nearly straight, .002 to .003 inches [50 to 76 microns] long; paraphyses filiform, numerous. Dead branches of leather leaf, *Cassandra calyculata*. Karner, N. Y. Aug.

Dr. H. D. House, State Botanist of New York, has kindly compared our specimens with Peck's type and regards them as the same.

We have found a few minor variations from Peck's description of the apothecial form. On the cranberry stem, as on Cassandra, the apothecia generally vary between 0.5 and 0.7 mm. in diameter, but on cranberry fruits they grow larger, in fresh specimens ranging from 0.5 mm. to 1.8 mm. with the majority 1 mm. or more in diameter. In our material, asci showed greater variation in length, measuring 58.8–117 by 5.0–7.7 microns. Ascospores in our specimens on Cassandra measured 55–74 by 2 microns, and on cranberry 39–74 by 1.5–3.8 microns, mostly 55–70 by 2–3 microns. Peck evidently considered the elongate, hyaline ascospores to be nonseptate, but we found that in mature condition they became clearly 4- to 7-septate, mostly 6- and 7-septate. Ascospore septation is also evident in stained sections of mature apothecia. Paraphyses often branch towards the apex. Figures 3 to 6, respectively, show the macroscopic appearance of apothecia on decayed cranberry, section of an apothecium on cranberry stem, a highly magnified portion of the hymenial layer, and mature and germinating ascospores. The end-rot fungus seems to be congeneric with the type of Godronia, *G. urceolus*, and should hereafter retain the name, *Godronia cassandrae* Pk.

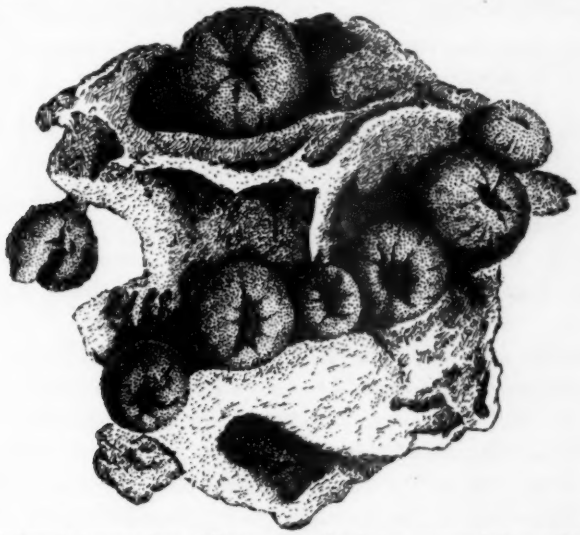

FIG. 3. *Godronia cassandrae*: Mature apothecia on mummied cranberry, X 15.

An examination of a specimen of *Dothiopsis myrtilli* Feltg. issued by F. Petrak in his Flor. Bohem. et Morav. Exs. No. 1609, as *Sirex cipulina myrtilli* (Oud.) Pet. shows that it is identical with *Fusicoccum putrefaciens* Shear. Petrak's specimens were found on *Vaccinium myrtilli* in Bohemia,

Apr. 30, 1923. Petrak (3), discusses this fungus and suggests that it may be the pycnidial form of *Godronia ericae* (Fr.) Rehm. This Godronia, however, is distinct from *G. cassandrae* Pk.

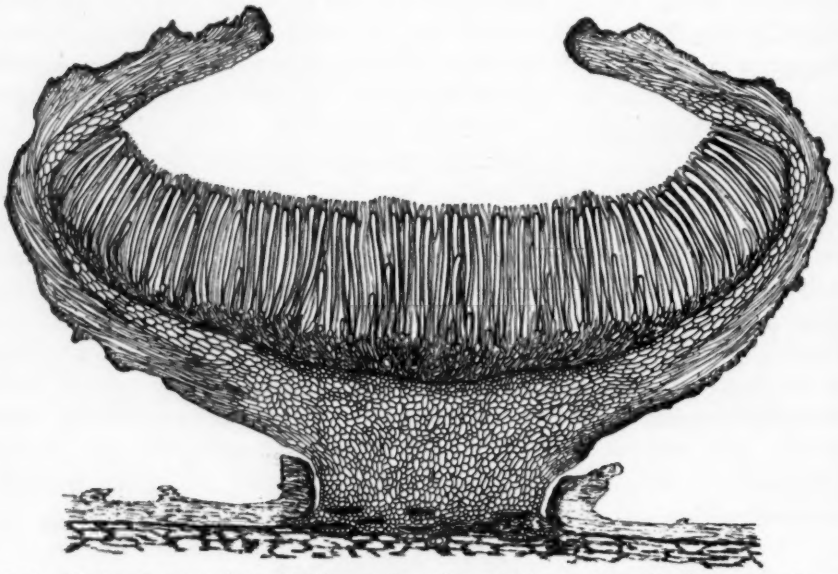

FIG. 4. *Godronia cassandrae*: Median section of apothecium cut longitudinal to cranberry stem, showing detailed structure of apothecium, X 137.

The apothetical form of *Godronia cassandrae* has been collected on stems and fruit of the cranberry in Massachusetts, Oregon, and Washington. Rudolph found one specimen on a fallen leaf in Washington. It has been found most frequently in the Pacific Coast region, where the climatic conditions seem to be more favorable for its development. As before stated, it has been collected also on Cassandra in New York and New Jersey.

Both pycnidia and ascomata of this fungus were collected also on dead twigs of *Vaccinum caespitosum* Mx. above timber line on Mt. Washington, N. H., July 12, 1922 (Shear and Stevens, Nos. 5159 and 5152).

A detailed description of the pycnidial form of the fungus *Fusicoccum putrefaciens* is not necessary here, as it has been described in detail elsewhere (5). Drawings of the pycnidia and pycnospores are reproduced in figures 1 and 2. The pycnidial form has been found on fruit and leaves of the cranberry, *Vaccinium macrocarpon* Ait., in New Jersey, Massachusetts, Michigan, Wisconsin, and Washington (5). In addition to collections on cranberry in the States named, Fusicoccum has been isolated from decaying berry tissue from the following hosts and localities: *V. macrocarpon,*

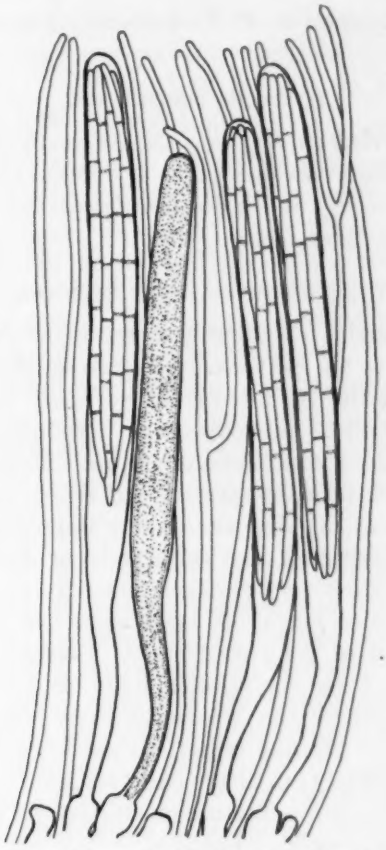

FIG. 5. *Godronia cassandrae*: Camera-lucida drawing of asci and paraphyses from section of apothecium on cranberry fruit, X 1000.

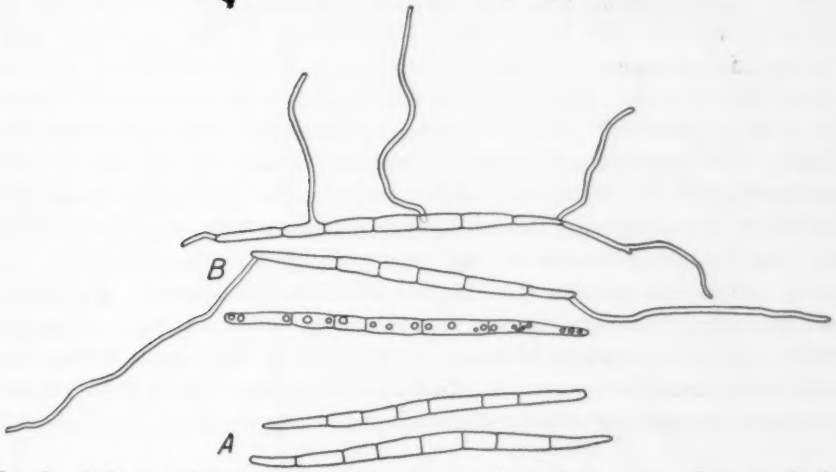

FIG. 6. *Godronia cassandrae:* A, mature, B, germinating ascospores, from apothecium on cranberry fruit, X 750.

North Carolina and Maine (8), *V. Vitis-Ideae,* Maine (8) and Alaska, and *V. oxycoccus,* Alaska (8).

ECONOMIC IMPORTANCE

Investigations carried on for the past ten years by Stevens, Rudolph, Franklin, and the present writers (1, 4, 6, 8, 9, 10) have shown that end rot causes greater loss to cranberries during storage, transportation, and distribution than any other factor.

THE PROBLEM OF BERRY INFECTION

End rot of cranberries is distinctly a storage trouble, as the rot rarely develops before berries are harvested. Unfortunately, the exact time and mode of infection of the fruit have not yet been definitely determined. Various difficulties involved in carrying out conclusive inoculation experiments with the fungus have not been overcome. Stevens (8) has shown in experiments carried on in Massachusetts that the end-rot fungus was present in young berries at the time the flowers were dropping. He isolated the end-rot fungus from such berries as early as July 6. Bain (1) also obtained the fungus from very young cranberries in Oregon on July 10, 11, and 14, in successive years. In most cases the fungus was found to be in the blossom end of the young berry. Stevens and Bain have also cultured the fungus from surface-sterilized living stems and leaves of cranberry in Massachusetts and Wisconsin, respectively. Isolations made from fruit later in the growing season do not show a much greater percentage of berries infected than at the early stage of the fruit. This would seem to show that infection either occurs during the blossoming period or immediately after, or is possibly systemic.

The fruiting forms of the end-rot fungus are relatively scarce, especially in Massachusetts, and it is difficult to account for the abundant infection of berries over large areas. The spores of the pycnidial form, Fusicoccum, are ejected and held together in glutinous masses not adapted to wind distribution. The ascospores may be disseminated in part by wind, but these are so rarely found in eastern cranberry bogs as to appear inadequate to produce the infection which usually occurs. Infection of the blossom and of the fruit may occur through the agency of insect pollinators of the cranberry blossom or may result from the flood waters which are frequently used in spring to protect the young growth from frost injury. It seems possible that there may be some systemic infection of the vines which may enter the fruit through its pedicel, as numerous cases of stem-end rot as well as blossom-end rot occur. Yet, the fact that considerable reduction of end rot is obtained by spraying with Bordeaux mixture could scarcely be explained except on the supposition that infection

for the greater part takes place through the blossom or surface of the fruit. Further investigation of these points of fundamental importance will be undertaken.

DEVELOPMENT OF END ROT IN THE BERRY

There is strong evidence that the end-rot organism develops rapidly only in berries which have been weakened by mechanical injury, partial smothering, senescence, or some other cause. The rot is extremely rare in berries before maturity and during the early period of storage, but, as has been repeatedly shown (4, 9, 10), it steadily increases as berries approach senescence. Shear, Stevens, Wilcox, and Rudolph (6) have demonstrated that injuries brought about by careless handling of berries during harvesting, screening, and packing operations lead to excessive development of the rot. As long as berries are provided with satisfactory storage conditions and are not too old, end rot develops but slowly; but, following mechanical injury or aging of the fruit, Godronia soon becomes the predominating cause of spoilage.

TEMPERATURE RELATIONS

Stevens, in 1917, presented data showing that *Fusicoccum putrefaciens* (*Godronia cassandrae*) in culture is able to grow slowly at a temperature of 0° C. (7). He pointed out at that time that this relation was favorable to the development of the fungus in the colder cranberry regions of the country. In a series of unpublished cold-storage experiments conducted in the fall of 1928, Stevens and Bain repeatedly isolated Godronia from berries which rotted at an accurately-controlled temperature of 0° C., thereby proving that the fungus is capable of destroying berries at that temperature. The ability of the fungus to grow at low temperatures is a factor of great importance in storing and marketing cranberries, since late berries are generally held at temperatures too low for the development of most other cranberry rots.

CONTROL METHODS

Two distinct methods of reducing loss from end rot are available. The first and most effective is by spraying. Numerous experiments in different parts of the country have shown that spraying with Bordeaux mixture two or three times during the summer gives satisfactory control of this as well as other storage rots (1, 5). The most important applications of spray are those made immediately preceding and following the blossoming period. The second method of reducing end rot involves regulation of storage and handling processes to check or delay the development of rot in infected berries. Factors known (6) to have an important bearing in this connection include (a) thorough drying of berries before storage; (b) adequate

ventilation; (c) prevention of unnecessary bruising of fruit; and (d) storage at low temperature, which, while not so effective as against other storage fungi having higher minimum-temperature requirements than *Godronia cassandrae*, is nevertheless of very definite value.[1]

SUMMARY

Evidence is presented demonstrating genetic relationship between *Fusicoccum putrefaciens* Shear, the cause of cranberry end rot, and *Godronia cassandrae* Peck, originally described on *Cassandra calyculata*.

The present known distribution and hosts of the fungus are given.

The pathology of cranberry end rot is briefly discussed, and control methods are outlined.

BUREAU OF PLANT INDUSTRY,
U. S. DEPARTMENT OF AGRICULTURE.

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10. ————, and ————. Storage rots of cranberries in the 1927 crop. Phytopath. **18**: 809–814. 1928.

[1] Since this paper was written, the junior author has made single ascospore cultures from *Godronia cassandrae* and obtained typical pycnidia of *Fusicoccum putrefaciens*, as found on cranberry, thus completing the evidence of the identity of the fungi on the two hosts.

AIRPLANE PHOTOGRAPHY IN THE STUDY OF COTTON ROOT ROT[1]

J. J. Taubenhaus, Walter N. Ezekiel, and C. B. Neblette

INTRODUCTION

Stakman and his associates used the airplane in their studies[2] on the spread of cereal-rust spores through the upper-air currents. They caught spores of several genera of pathogenic fungi, conidiophores, pollen grains, glumes of grasses, and small insects on microscope slides exposed at elevations varying from 1,000 to 16,500 feet. Airplanes are used extensively in dusting for various insects and have been tried for plant diseases. So far as the writers are aware, however, no previous studies of plant diseases have been made by airplane photographs.[3]

DIFFICULTY IN PHOTOGRAPHING ROOT ROT

Cotton root rot, caused by the fungus *Phymatotrichum omnivorum* (Shear) Duggar, is one of the most serious plant diseases of the Southwest and entails annual losses running into millions of dollars. The disease causes definite dead areas or spots in the fields. These spots stand out prominently and are conspicuous even from considerable distances, as when viewed from moving trains. Yet, it always has been difficult or impossible to obtain satisfactory photographs showing the relative severity of root rot in naturally-infected fields or in experimental plots.

No matter how few or how many plants are killed by root rot in a given field, there is no general mass effect visible unless nearly 100 per cent of the plants are killed. In a field with 50 per cent loss from root rot, the remaining 50 per cent of the plants are as large as they would be were all the plants sound. Similarly, photographs of plants that have died from root rot in fields where nearly every plant has been killed would apply just as well to the occasional dead plants in fields where losses do not exceed 2 per cent.

With photography from the ground, the inevitable foreshortening in the picture emphasizes the few plants in the foreground and the comparative value of the photograph is based on these plants. Photographs are some-

[1] Published with the approval of the Director as contribution No. 462 of the Texas Agricultural Experiment Station.

[2] Stakman, E. C., Henry, A. W., Curran, G. C., and Christopher, W. N. Spores in the upper air. Journ. Agric. Research, 24: 599–606. 1923.

[3] This work has been mentioned already as follows: Neblette, C. B. Aerial photography for study of plant diseases. Photo-Era Mag. 58: 346. 1927. Airplane photography for plant disease surveys. Photo-Era Mag. 59: 175. 1928.

times obtained in which a few surviving, normal plants in the foreground obscure the great mass of dead plants even in fields in which almost every plant has been killed. Again, where root rot is scarce, it may either be overlooked in the photograph, or the picture may give the impression that the disease has involved most of the plants in the field. For comparative purposes, photographs must therefore be general views which include entire plots or fields and at the same time differentiate plants killed by root rot from the normal plants. This has been done with reasonable satisfaction by ordinary photography only for experimental plots of single rows.

It may be noted that this optical obscurity of losses from root rot is a hindrance also to rapid visual estimation of losses. When root rot occurs in more than one spot in a field estimates of losses are nearly always too low since the surviving plants are so much more conspicuous to the eye than those plants that have wilted.

On numerous occasions army fliers stopping over at College Station reported to us that root rot spots were readily visible from the air, even when flying at high altitudes. This suggested that root rot spots might perhaps be photographed more successfully from an airplane. One of us (Neblette) has made such photographs. The method appears so valuable as to suggest its use in furnishing estimates of losses as well as in furnishing better pictures of the results of field experiments with root rot.

AERIAL PHOTOGRAPHIC METHODS

Several test flights were made in October, 1927, near Substation No. 5 at Temple, where root rot is widespread. Some of the photographs secured at that time are shown in figure 1. Subsequent flights were made from Temple to Waco, a distance of seventy miles. It was found that the best times of day to make these exposures were at 10 A. M. and 3 P. M., or at other times when the sun was shining brightly. Results were not so good when exposures were made during cloudy weather or at noon because heavy shadows cast by the leaves of normal plants help to differentiate them from plants with root rot. The height varied from 250 to 500 feet. An ordinary Graflex camera, size 5 x 7, fitted with a Bausch & Lomb f 4.5 Tessar lens of 10-inch focus, was used with Eastman commercial panchromatic film and a K1 (light yellow) filter. An aero filter made especially for aerial photography probably would have given sufficient color correction to eliminate the usual slight atmospheric haze with less increase of exposure.

Ordinary hand or Graflex cameras are not used customarily in aerial photography, so it may be of interest to give the methods. It must, of course, be remembered that the camera is being used for purposes for which it was not designed and one must be prepared to accept larger percentages of failures, but comparatively good pictures may be secured by proper

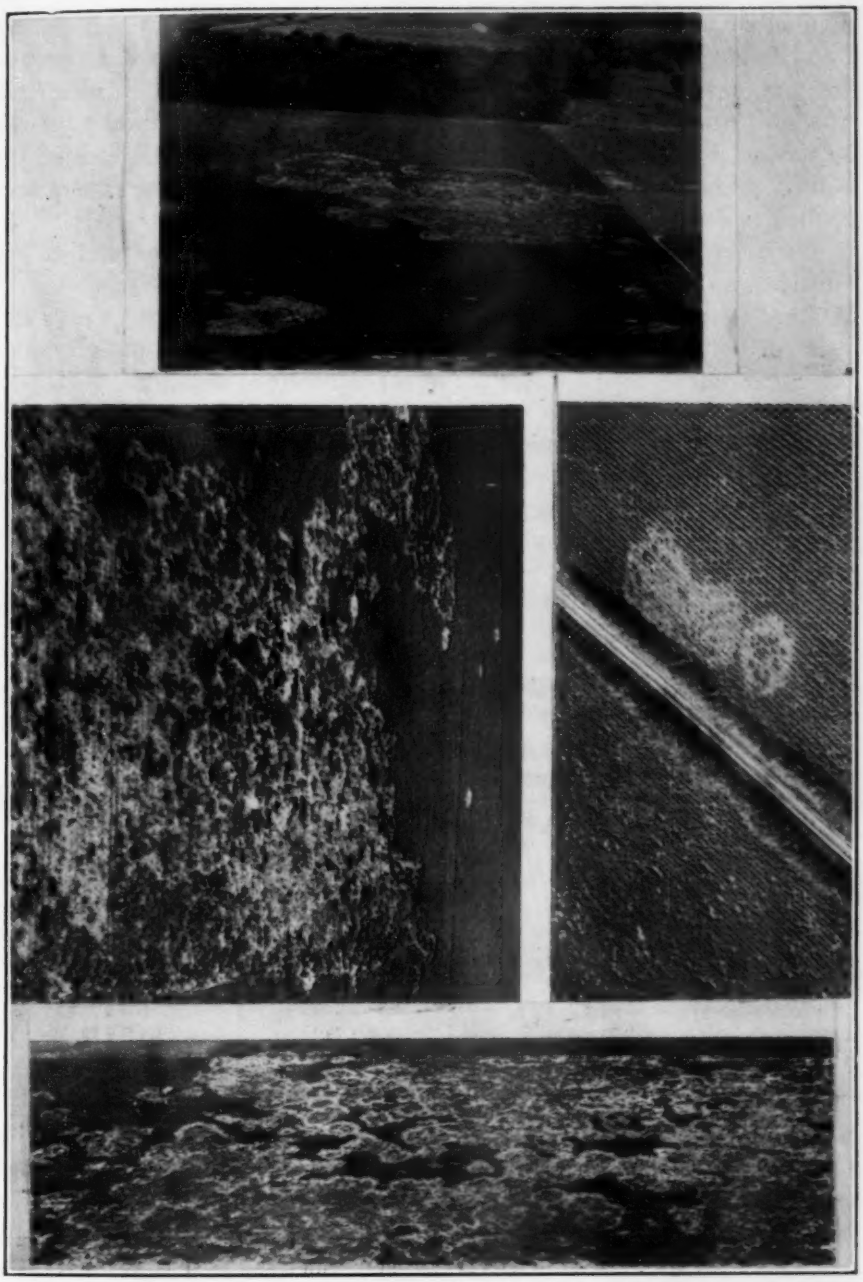

FIG. 1. Airplane views of four cotton fields showing root rot spots of various sizes and shapes. The dark regions in the photographs are areas of normal plants, and the light regions are the dead spots.

manipulation. As it is difficult to focus the camera while in the plane, the focus should be accurately adjusted to infinity before one leaves the ground and the lens secured in such fashion that the focus cannot be disturbed. The hood provided for focusing must be closed down as it would otherwise be torn off by the wind when the camera is held out over the side of the plane. A small direct vision finder attached to the side of the camera may be used to determine the area included in the picture.

Ordinary film may serve in clear atmospheric conditions and at low altitudes up to 500 feet. However, since panchromatic films may be obtained now not only in professional sized flat films, but also in rolls and packs of the usual sizes, there is no necessity for using any other type of film regardless of the requirements of the camera one is using. With panchromatic film an Aero 1 or a Wratten K1 filter is sufficient to give excellent contrast under usual conditions.

The elevation at which pictures are to be made depends on the purpose of the work, the focal length of the lens and the permissible exposure. The area covered in a single photograph will depend on the altitude at time of exposure, the focal length of the lens, and the angle of view in the case of oblique views. Table 1 gives the areas included in photographs at different altitudes.

TABLE 1.—*Dimensions of field included in vertical photographs with lenses of 6, 8, and 10 inches equivalent focal lengths at various altitudes*[4]

Altitude, feet	Dimensions of field included in photographs, feet		
	6 inch lens with 4 x 5 plate	8 inch lens with 5 x 7 plate	10 inch lens with 7 1/16 x 9 1/2 (18 x 24 cm.) plate
250	60 x 71	100 x 122	175 x 225
500	120 x 142	200 x 244	350 x 450
750	180 x 213	300 x 376	525 x 675
1000	240 x 284	400 x 488	700 x 900
1250	300 x 355	500 x 610	875 x 1125
1500	360 x 426	600 x 732	1050 x 1350
2000	480 x 568	800 x 976	1400 x 1800

While fairly sharp results may be obtained in aerial photography with a Graflex camera or with a focal-plane hand camera of the type represented by the Cycle Graphic, more accurate work would require a camera especially

[4] This table has been calculated for use with hand cameras and reflex cameras, such as the Graflex, at lower elevations than is customary with standard aerial cameras. It is not advisable to attempt to work with ordinary cameras from the high altitudes commonly used in survey work with aerial cameras.

designed for aerial photography. A comparatively wide range of such cameras is now available. Fully-recording mapping cameras such as the Fairchild require special installation in the plane and the price is prohibitive for all but those commercially engaged in aerial photography. The Folmer type K-5 camera is less expensive, but the other objection mentioned still holds. Both the Fairchild and the Folmer cameras use Eastman aerial film which is supplied in rolls 25 cm. (9½ inches) wide for 37, 70, or 100 exposures, 18 x 24 cm. An excellent camera for general airplane photography at a more moderate cost is the Folmer type A-1, making pictures 4 x 5 inches in size. This type of camera may be used for both oblique and vertical pictures but is not suitable for exact mapping purposes. It is possible to build a satisfactory camera of this type for considerably less cost than the commercial article, purchasing the lens and a suitable focal-plane shutter. Such a 5 x 7 camera built to the specifications of one of the writers is to be tested during the coming summer and fall.

SUMMARY

(1) Damage caused by cotton root rot is such as to make it difficult or impossible to secure from the ground photographs that show satisfactorily relative losses in fields or in plots of more than a single row.

(2) Airplane photographs have solved the problem by furnishing views which include entire fields and at the same time differentiate plants killed by root rot from the normal plants.

(3) Suggestions are given on airplane photography with ordinary hand cameras.

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A NEW HOST OF SUGAR BEET CURLY TOP

RUTH COLVIN STARRETT

INTRODUCTION

In a publication by Carsner[1] the following families have been reported to be susceptible to curly top: Amaranthaceae, Caryophyllaceae, Chenopodiaceae, Geraniaceae, Leguminosae, Malvaceae, Polygonaceae and Urticaceae. Severin and Henderson[2] have mentioned three families to be naturally infected with curly top, namely, Chenopodiaceae, Leguminosae and Cucurbitaceae.

Oxalis stricta L., according to the International Code of Classification belongs to the Geraniaceae family but the more recent edition of Gray, and also Britton and Brown, have assigned it to a new family, Oxalidaceae. The common occurrence of Oxalis and its very high acidity make it an interesting addition to the list of susceptible plants. The acidity for normal leaves and young stems is pH 2.45, but for the young leaves it is pH 2.23.[3]

GREENHOUSE EXPERIMENTS

In the investigations carried on at Arlington, Virginia, during the year 1928, experiments were made to determine the relationships of curly top in *Oxalis stricta* and sugar beets, and the ability of *Eutettix tenellus* (Baker) to transmit this disease from Oxalis to Oxalis, from Oxalis to beet, and from beet to Oxalis.

The first diseased plant of *Oxalis stricta* appeared as a volunteer growing in a pot along with a caged beet plant affected with curly top. This Oxalis plant had pronounced papillae arranged along the veins on the under side of the leaves varying in size and shape as in curly top of sugar beet. The entire plant was distorted in appearance due to the curled and twisted leaves and the reaction to infection was so severe that no normal leaves developed (Fig. 1, A). It remained green in color but considerably dwarfed, although the presence of the disease did not interfere with the regular seed production.

Three types of transmission experiments were tried, then each one repeated entirely with confirmatory results. One table for each type of experiment is given in this paper to demonstrate the results.

[1] Carsner, E. Susceptibility of various plants to curly-top of sugar beet. Phytopath. 9: 413–421. 1919.

[2] Severin, H. H. P., and C. F. Henderson. Some host plants of curly top. Hilgardia 3: 339–384. 1928.

[3] The writer is indebted to Dr. A. M. Hurd-Karrer for the pH determinations.

FIG. 1. Plants used in experiments 1 and 2. A. Typical effects of curly top upon the leaves of *Oxalis stricta*. B. Sugar-beet plants with typical curly-top symptoms produced by leaf hoppers previously fed upon the diseased Oxalis leaves, shown in A. C. Oxalis plants in which curly top was reproduced by leaf hoppers fed on the sugar-beet plants, shown in B.

In order to determine if the disease were curly top the plant was isolated in a large cage and nonviruliferous individuals of *Eutettix tenellus* were allowed to feed on it. After a week of uninterrupted feeding, these leaf hoppers were removed to small leaf cages by the suction-pipe method, two insects in each cage, and attached to healthy leaves of young sugar-beet seedlings. Two days were allowed for feeding on the beet plants and the leaf hoppers were then returned to the original Oxalis plant. Curly-top symptoms appeared in twenty-four days on the sugar beets and quickly became severe (Fig. 1, B, Table 1).

TABLE 1.—*Transmission of curly top of Oxalis to sugar beet by Eutettix tenellus*

Beet seedling no.	No. of insects	Results			
		11–30–28	12–7–28	12–10–28	1–11–29
		Date of inoculation Nov. 6, 1928			
I	2	Normal	Normal	Normal	Normal
II	2	Smallest leaves veinlets cleared	More decided clearing	Definite case of curly top	Positive case
III	2	?	Decided veinlet clearing	Positive curly top	Positive case
IV	2	Normal	Normal	Normal	Normal
	Checks				
I	2	Normal	Normal	Normal	Normal
II	2	Normal	Normal	Normal	Normal

In order to complete a cycle of transmission, nonviruliferous leaf hoppers were caged on one of the beet seedlings that had developed a typical case of curly top (Table 1) and, after the regular time allowed for feeding, were removed to four healthy Oxalis plants. Three of these plants developed unmistakable signs of the disease in about one month (Fig. 1, C) (Table 2).

Leaf hoppers were then placed on one of the curly-top Oxalis plants and later transferred to four healthy Oxalis plants for two days, four viruliferous leaf hoppers in each large cage. Two of these plants showed definite signs of curly top in about eighteen days (Table 3).

Because of the reported transmission of a disease of sugar beet resembling curly top in Argentina by leaf hoppers of the genus Agallia, two species that had been found to feed on beets at Arlington, *A. constricta* and *A. sanguinolenta,* were also used in these experiments.

All the conditions were duplicated, the periods of feeding being exactly parallel with those used for *Eutettix tenellus,* greenhouse conditions were

TABLE 2.—*Transmission of curly top of beet to Oxalis by Eutettix tenellus*

Date of inoculation Dec. 10, 1928					
Oxalis stricta no.	No. of insects	Results			
		1–7–29	1–12–29	1–18–29	1–21–29
I	4	Small leaf veinlets cleared	Definite curly top	Curly top	Curly top
II	4	Normal	Normal	Normal	Normal
III	4	One leaf with papillae	Definite case	Curly top	Curly top
IV	4	Decided papillae	Definite case	Curly top	Curly top
	Checks				
I	4	Normal	Normal	Normal	Normal
II	3	Normal	Normal	Normal	Normal

the same, and both Oxalis and beets were used as host plants, but in each experiment the results were negative.

TABLE 3.—*Transmission of curly top from Oxalis to Oxalis by Eutettix tenellus*

Date of inoculation Jan. 15, 1929				
Oxalis stricta no.	No. of insects	Results		
		1–26–29	2–2–29	2–5–29
I	4	Young leaf slightly curled	Positive curly top	Positive curly top
II	4	Normal	Normal	Normal
III	4	Suspicious	More definite markings	Positive curly top
IV	4	Normal	Normal	Normal
	Checks			
I	7	Normal	Normal	Normal
II	10	Normal	Normal	Normal

SUMMARY

The results of the experiments prove that curly top can be carried from *Oxalis stricta* to beet and inversely through the agency of *Eutettix tenellus.*

Curly top, in Oxalis, checks the growth of the plant but never results in a total destruction, even in cases of severe infection.

In Oxalis, curly top usually is manifest in a conspicuous development of protuberances or papillae on the under surface of the leaves.

Two species of Agallia, *A. constricta* and *A. sanguinolenta,* were tested as vectors with negative results.

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STORAGE ROTS OF CRANBERRIES IN THE 1928 CROP

NEIL E. STEVENS AND HENRY F. BAIN

During each of the past three years the writers have attempted to measure quantitatively the actual storage rot caused by various fungi in cranberries in commercial storage. The chief interest attaches, of course, to a comparison of the results obtained in different years, from different regions, and with different varieties. Where there are so many variable factors, the results cannot be made strictly comparable, yet they do permit of some comparison and in many ways are more interesting than if artificial conditions, such as constant storage temperatures, had been maintained. The most serious break in our records for this year is that, largely because of the very early harvest in that region, no Oregon or Washington berries were included in the storage test.

METHODS

The methods followed were substantially those of 1926 and 1927,[1,2] fully described in earlier papers. The chief difference, other than the omission of berries from the West Coast, was that the berries had to be stored in the third story of the building in Chicago; whereas, in 1926 and 1927, they had been kept in the basement. As the windows of this storeroom were open, the berries were subjected to much lower temperatures in October, 1928, than in the same month in 1926 or 1927 (Table 1). On the other hand, the November temperatures of 1928 corresponded very closely to those of 1926, and the December temperatures for 1928 were well above either 1926 or 1927.

TABLE 1.—*Approximate Storage Temperature, Chicago, in Degrees F.*

	1926	1927	1928
October	65–55	70–60	62–46
November	53–43	58–48	55–44
December	40–30	48–35	50–40

RESULTS

The results are shown graphically in figure 1. Comparing the keeping quality of the stored lots for the three years, it is evident that, for Wisconsin, the keeping quality of the Howes was better in 1927 than in 1928 (no

[1] Stevens, N. E., and H. F. Bain. Storage Rots of Cranberries in the 1926 Crop. Phytopathology, 17: 649–665, 1927.

[2] ———, ———. Storage Rots of Cranberries in the 1927 Crop. Phytopathology, 18: 809–814, 1928.

Wisconsin Howes were included in the 1926 test), and for McFarlins, better in 1927 than in either 1926 or 1928. For Massachusetts, both varieties under test showed the best keeping quality in 1926 and the poorest in 1927, while for New Jersey, both varieties were poorest in 1927 and best in 1928.

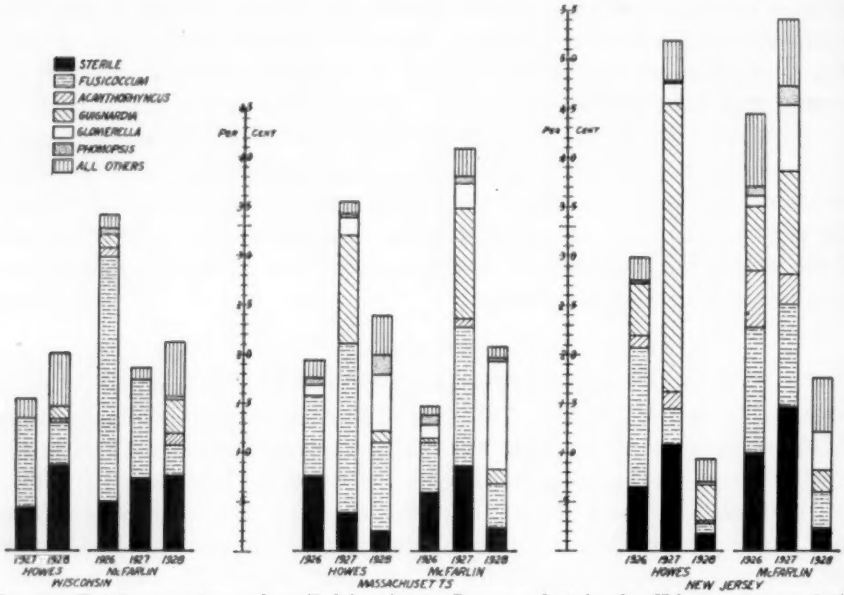

FIG. 1. Total percentage of spoiled berries to January first in the Chicago storage tests for the crops of 1926, 1927, and 1928, with proportions initiated by different fungi and sterile breakdown shown by suitable conventions.

It can not be assumed that the berries used in these storage tests are always representative of the crops of the various regions. For example, the Massachusetts Howes used in 1928 were somewhat too green when harvested to show the best keeping quality or to fairly represent the Massachusetts crop of that year. In some cases, however, the results shown in figure 1 seem to agree very well with what is known of the keeping quality of the entire crop of their respective regions. The results for New Jersey, for example, show a surprising agreement with a keeping quality index worked out by a wholly different method, that is, the comparison of the market claims allowed for decay in 1926, 1927, and 1928.[3] All observations agree in showing that the keeping quality of the crop of the different regions varies independently.

[3] Stevens, N. E. Six Years' Experience in Forecasting the Keeping Quality of the Cranberry Crop in Wareham and Carver, Massachusetts. Proceedings American Cranberry Growers' Association, 59: 8–16, 1929.

COMPARATIVE KEEPING QUALITY OF THE HOWES AND MCFARLIN VARIETIES

Special importance is attached at this time to a comparison of the keeping quality of the Howes and McFarlin varieties. The Howes has long been recognized as the standard late variety of cranberry. The McFarlin is the most important of the so-called fancy or large size cranberries. It has come into prominence during the past few years because of its very high degree of resistance to the destructive false-blossom disease.

A careful examination of figure 1 shows that, for any given region and year, the keeping quality of the McFarlin up to January first is only slightly inferior to that of Howes. In Massachusetts, indeed, McFarlins were better both in 1926 and 1928 than the Howes. The superior keeping quality of the Howes variety is shown more markedly during January and February, but in many years a very large proportion of the crop is marketed before January.

Regarding the importance of the different fungi, the only outstanding variation from earlier years is the large percentage of Glomerella in both Howes and McFarlin in the Massachusetts crop. It may be noted that the amount of sterile breakdown is very low in both varieties for Massachusetts and New Jersey in 1928.

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WASHINGTON, D. C.

STEM RUST OF WHEAT IN PERU[1]

E. V. Abbott

Stem rust is the most important disease of wheat in Peru. It occurs every year in epidemic form in many of the highland valleys of the mountains where this crop is grown extensively and, on the coast, it is so destructive that wheat is no longer cultivated there. It is doubtful whether wheat could compete with either cotton or sugar cane as a major crop on the coast, even if a rust-resistant variety were available, but it could supplement them and find large use as a food for livestock if not for human consumption. The same is true of barley, the cultivation of which is at present limited to the mountain valleys because of rust.

In an effort to obtain a rust-resistant wheat for the coast as well as the highlands, sixty varieties were imported for field study at the Lima experiment station during 1928, the seed of which was furnished through the courtesy of the United States Department of Agriculture and several of the State experiment stations in the United States. Both common and durum wheats were included, as well as two emmers.

The first planting was made in rows one meter apart, the middle of June, which, in Peru, is the beginning of the winter season. A second planting of several of the varieties was made in August. Inasmuch as rain does not fall on the Peruvian coast, water was supplied by irrigation. During the period from June to October heavy fogs and mists are very common at Lima, but they do not furnish sufficient moisture for crop growth.

The results obtained with eleven of the imported varieties, which are representative of the sixty tested, and one native wheat, are briefly summarized in table on the following page.

It will be noted that Pusa-4 showed the lowest percentage of infection among the common wheats and that it matured grain. This was due entirely to its earliness and consequent rust escape rather than to rust resistance. The same variety planted in August rusted so heavily that not a single kernel matured, which was also true of the other common and the durum varieties.

The outstanding feature of the results and the point of most importance are the high degree of infection of Khapli emmer. In the June planting of this variety, infection by stem rust was 50 per cent, with the degree of infection of individual plants heavy, while in the August planting 100 per cent of the plants rusted, the degree of infection being classed as very heavy. The heads produced from the late sowing were smaller than normal and the grain was slightly shrivelled.

[1] Contribution from the Experiment Station of the National Agrarian Society, Lima, Peru. Published with the approval of the Director.

TABLE 1.—*Infection of wheat varieties by Puccinia graminis tritici (Pers.) Eriks. and Henn.*

Variety	Per cent plants rusted[1]	Degree of infection[2]	Grain matured
1. Little Club	100	Very heavy	No
2. Hope	100	Heavy	Yes; grain slightly shrivelled
3. Kanred	100	Heavy	Yes; grain shrivelled
4. Marquis	100	Very heavy	No
5. Monad	100	Very heavy	No
6. Pentad	100	Very heavy	No
7. Pusa-4	88	Medium	Yes; grain normal
8. Webster	100	Very heavy	No
9. White Federation	100	Very heavy	Few plants matured
10. Native common wheat	100	Very heavy	No
11. Khapli emmer	50	Heavy	Yes; grain normal
12. Yaroslav emmer	93	Heavy	Yes; grain slightly shrivelled

[1] Based on counts of 500 to 1000 plants.

[2] Infection of individual plants, based on number and size of pustules.

The high degree of infection of Khapli indicates the presence of a new physiologic form of *Puccinia graminis tritici* in Peru. According to the key of Stakman and Levine[2] for the identification of physiologic forms of this fungus, the Peruvian form would be form XV; but in view of the great difference in the percentage of infection Khapli in Peru and the maximum previously reported,[3] it seems probable that they are not identical. Infection experiments employing the twelve differential hosts of Stakman and Levine have not yet been completed. Studies are being made to determine whether there are morphologic differences between the Peruvian form and form XV.

In considering the problem of stem rust epidemics in Peru the questions naturally arise as to how the causal fungus survives from one crop season to the next, and what rôle species of Berberis may play in dissemination of the disease. As opportunities have arisen from time to time, observations have been made in the wheat-growing valleys of the mountains of Peru with these questions in mind.

Species of Berberis are common in the Peruvian Andes, seven members of the genus being found there, according to Weberbauer.[4] The writer has

[2] Stakman, E. C., and M. N. Levine. The determination of biologic forms of *Puccinia graminis* on *Triticum* spp. Minn. Agr. Exp. Sta. Tech. Bul. 8. 1922.

[3] Clark, J. A., J. H. Martin, and E. C. Stakman. Relative susceptibility of spring-wheat varieties to stem rust. U. S. Dept. Agr. Dept. Circ. 365. 1926.

[4] Weberbauer, A. Die Vegetation der Erde. XII, Die Pflanzenwelt der peruanischen Anden. Leipzig, 1911.

observed two species of barberry growing along the roadsides bordering fields of rusted wheat and on the surrounding hillsides in great abundance near Arequipa, Cuzco, Huancayo, Jauja, and Tarma, but in no instance were they infected with *Puccinia graminis*. The elevations of these valleys vary from nine to thirteen thousand feet above sea level. The observations thus far made are too limited, however, to permit the conclusion that the barberry plays no part in the life cycle of stem rust in Peru.

Another point not yet determined is whether the urediniospores of the fungus are able to survive from one crop season to the next in the same locality in which they are produced. It is possible that at high altitudes they do not survive the cold and that, on the coast, the high temperatures destroy their germinability. But, even though they be not killed by unfavorable environmental conditions in any given locality, or though there be no alternate host present on which they may pass the intervening period, there is such a succession of crops at the different altitudes in Peru that it seems probable the fungus might easily live continuously on wheat, merely passing from one district to another. Throughout the entire year there is not a lapse of more than about three months when wheat is not growing somewhere in the mountain valleys, the stage of development varying gradually with the differences in elevation. In view of what is known regarding the carrying of rust spores by air currents, it is not at all difficult to conceive of the fungus living continuously on wheat and being carried from one section to another by the wind. This appears to offer the only probable explanation of what occurred at Lima in 1927 when experimental sowings of wheat were made near Lima by the Federal Department of Agriculture. So far as is known, this was the first time in many years that wheat had been planted in this region. A heavy and general infection by *Puccinia graminis tritici* resulted, the disease appearing almost simultaneously in all parts of the field of about two hectares in size. There is a possibility that the fungus hibernates in this section on some wild grass, but no such host has yet been discovered. Wind-blown spores from the mountains seem to offer the most plausible explanation of these results, as well as those obtained at the Experiment Station in 1928.

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RELATION OF TIME OF ROGUING TO THE SPREAD OF SPINDLE TUBER IN SEED POTATO PLATS[1]

H. O. WERNER

Early roguing of potato seed plats is commonly advised as a means of controlling virus diseases, since by removing the source of infection early in the season, there is supposedly less opportunity for disease to be transmitted to healthy plants. Some data upon the effect of early caging and early harvesting in relation to the spread of spindle tuber were presented in an earlier paper. It has also been shown that spindle tuber spreads more rapidly under irrigation than on dry land, and under eastern or central Nebraska early crop conditions than under the late crop conditions in the western dry-land areas, Werner.[2] Therefore, it seemed advisable to determine the extent to which spindle tuber can be controlled by the early roguing of seed plats planted early, under irrigation, at North Platte.

EXPERIMENTAL PROCEDURE

In April, 1925, spindle-tuber[3] Triumph potatoes were planted under irrigation at North Platte, Nebr., in every 5th hill in rows of certified Irish Cobbler potatoes, thus providing 20 per cent of known original infection. The Irish Cobbler seed had been grown on dry land in northeastern Wyoming and contained less than 1 per cent of spindle tuber. Triumph spindle-tuber potatoes were used because of the ease of distinguishing them from the Irish Cobbler, thus permitting the definite separation of the spindle tubers in the Irish Cobbler strain from the spindle-tuber Triumphs inserted as sources of infection. Schultz and Folsom[4] found that the spindle-tuber disease is not altered by transmission from one variety to another, and also that intervarietal transmission occurs as readily as intravarietal transmission. About 50 per cent of the plants had emerged by May 25. The Triumph spindle-tuber plants were removed from certain blocks (3 adjacent rows) at three-week intervals, beginning June 10.

The plats reserved as controls, in which no spindle tuber was planted, were hardly reliable control plats, since they were in the same general

[1] Published with the approval of the Director as paper No. 75 Journal series, Nebraska Agricultural Experiment Station.

[2] Werner, H. O. The spindle-tuber disease as a factor in seed potato production. Nebr. Agr. Exp. Sta. Res. Bul. 32. 1926.

[3] In this paper no effort is made to differentiate between spindle tuber and unmottled curly dwarf, because when the experiment was started the symptom differences were not clearly understood.

[4] Schultz, E. S., and Donald Folsom. Transmission, variation, and control of certain degeneration diseases of Irish potatoes. Jour. Agr. Res. 25: 43–117. 1923.

area, and the outside rows of these plats were adjacent to other experimental lots. A more satisfactory control was provided in a larger irrigated planting of this stock which was made in an isolated plat in a corn field.

The Irish Cobbler tubers produced in these plats were weighed according to grade. Special attention was paid to the prevalence of spindle tubers in each lot of potatoes. The spindle-tuber Triumphs produced in these plats were discarded at harvest time. Irish Cobbler seed potatoes were selected at random from the center row of each plat for planting in comparative trials under irrigation in 1926. In these trial plats there were duplicate plats of 3 rows each, the rows being 3 rods long.

At harvest time the tubers were carefully graded by size and all tubers of No. 1 size, showing definite spindle-tuber symptoms, were sorted out and weighed separately. This same procedure was duplicated in another experiment conducted during the years 1926 and 1927. The results for both experiments are reported and discussed together, because the results are fundamentally the same.

EXPERIMENTAL RESULTS

Seed plats. There was no evidence in either year of any current season spindle-tuber infection in the Irish Cobblers, due to the interplanting of spindle-tuber Triumphs. The plants and tubers were examined critically for any evidence of spindle tuber. The spindle-tuber readings reported in table 1 indicate that about the same percentage of spindle tuber was produced in the Irish Cobbler plats, into which no spindle-tuber Triumphs were planted, as in these plats from which the inserted Triumphs spindle-tuber plants were never removed. Undoubtedly the spindle-tuber potatoes sorted out were produced by plants grown from infected seed rather than as a result of current-season infection. There was much variation in yield from the different seed plats but none of this could be attributed to the inserted spindle tubers.

Comparative plats. When the tubers produced in the seed plats were tested in the comparative trial plats the following year it was found that when roguing of the seed plats had been delayed beyond June 10, there was a marked decline in yield and a great increase in the percentage of spindle tubers and small potatoes (Fig. 1 and Table 1).

The total yield from the various lots in the comparative plats decreased as the removal of the spindle-tuber plants had been delayed the previous year (Fig. 1, A). The differences in yield, due to roguing date, were more pronounced when only No.-1-size healthy tubers were considered.

Spindle tuber was responsible for the production of many small tubers. A positive correlation existed between the percentage of No.-1-size spindle tubers and the percentage of No. 2 and No. 3-size tubers.

TABLE 1.—*The effect of interplanting healthy Irish Cobbler seed potatoes with spindle-tuber Triumphs (rate of 4 to 1) upon yield and percentage of spindle tuber during the year seed tubers were produced, and the following year when grown in comparative plats. Seed plats and comparative tests planted under irrigation.*

A. Experiment conducted in 1925 and 1926

Treatment in 1925	Total yield, bushels per acre		Spindle tubers of No. 1 size—per cent of total yield	
	1925 (Seed plat)	1926 (Comparative plat)	1925 (Seed plat)	1926 (Comparative plat)
Check (grown in Wyoming on dry land)	*	346	*	1.2
No spindle-tuber Triumphs inserted in isolated plat	*	259	*	14.7
No spindle-tuber Triumphs in nonisolated plat	450	304	2.8	22.4
Triumph spindle-tuber plants removed June 10	390	264	0.5	26.3
Triumph spindle-tuber plants removed July 1	408	244	1.9	52.0
Triumph spindle-tuber plants removed July 20	414	202	1.2	63.5
Triumph spindle-tuber plants removed Aug. 10	550	177	1.1	61.4
Triumph spindle-tuber plants not removed	498	160	2.3	59.7

* Data not recorded in these seed plats.

B. Experiment conducted in 1926 and 1927

Treatment in 1926	1926 (Seed plat)	1927 (Comparative plat)	1926 (Seed plat)	1927 (Comparative plat)
Check (grown in Wyoming on dry land)	*	322	*	1.1
No spindle-tuber Triumphs inserted in isolated plat	*	307	*	1.1
No spindle-tuber Triumphs in nonisolated plat	346	209	1.5	4.6
Triumph spindle-tuber plants removed June 10	283	194	1.5	4.1
Triumph spindle-tuber plants removed July 10	303	88	2.0	32.4
Triumph spindle-tuber plants removed July 20	340	56	1.6	35.7
Triumph spindle-tuber plants removed Aug. 10	219	112	2.4	19.9
Triumph spindle-tuber plants not removed	269	127	1.6	8.7

* Data not recorded in these seed plats.

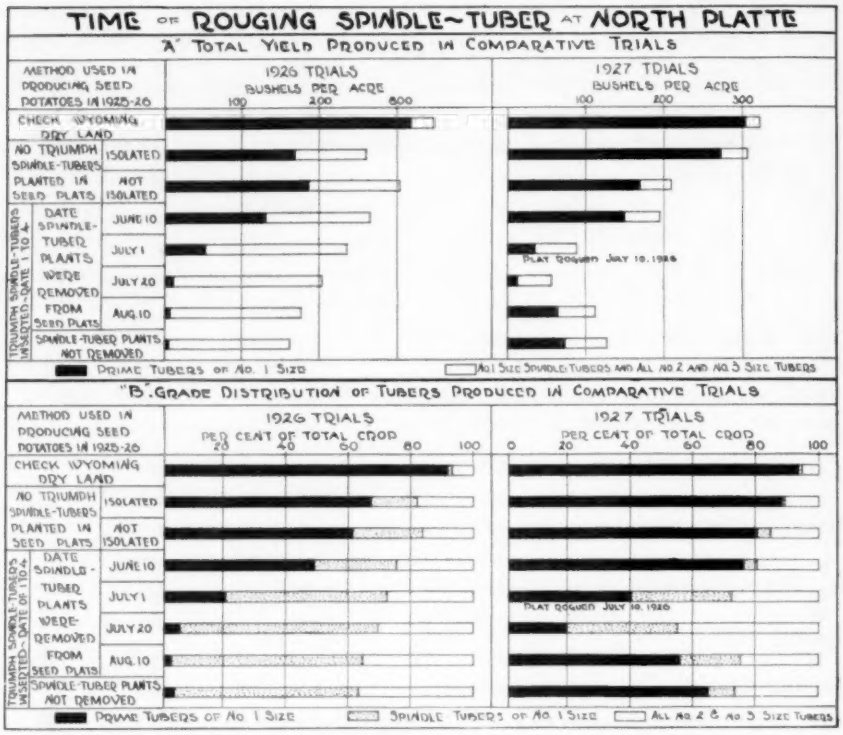

FIG. 1. The effect of the time of roguing interplanted spindle-tuber Triumphs from Irish Cobbler seed plats in relation to yield, grade, and spindle-tuber content the following season. Dates refer to years of comparative trials. Each year a new line of Irish Cobbler seed was planted in the seed plats.

In 1925 conditions were more favorable for transmission than in 1926. This accounts for the percentage of spindle tuber being greater in the 1926 than in the 1927 comparative plats, as shown by comparable lots. In 1926 the plants in the plats rogued July 10, July 20, and part of the August 10 plat remained green until harvest time in September; whereas, the plants in the other plats matured about the middle of August, due to accidental local soil-moisture shortage. The result of this difference in maturity was seen in the spindle-tuber content and yield in the 1927 comparative plat. The early maturing plats—even though not rogued or rogued late—produced seed with less spindle-tuber infection than did the early-rogued plats in which the plants remained green till the end of the season.

CONCLUSIONS

Spindle-tuber Triumphs, interplanted in seed plats among healthy Irish Cobblers (1 to 4), were rogued out at intervals of approximately three weeks.

Under the conditions of this experiment, *i.e.*, the production of seed potatoes as an early crop (April to September) under irrigation at North Platte, there were no current-season symptoms of spindle-tuber infection in either the plants or tubers produced in these seed plats; neither was the current-season yield appreciably affected. However, the comparative plantings the following year indicated that considerable infection had occurred as manifested by a high percentage of spindle tuber and reduced yields. Roguing done June 10 (7 weeks after planting) was reasonably effective but roguing after that date was of little value. In this experiment we knew the exact location of the diseased plants which were to be rogued. Such precise practice is not possible in commercial fields. Early maturity due to drought was quite effective in preventing the spindle-tuber virus from reaching the seed tubers.

The presence of a small amount of spindle tuber in Irish Cobblers did not always markedly diminish the total yield, but it did reduce the amount of primes by reducing the size of the tubers and by increasing the number of rough, bulging-eye tubers, thus detracting from their appearance and market value.

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COLEODICTYOSPORA, A NEW GENUS OF DEMATIACEAE

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In examining some diseased sugar-cane material from Cuba, collected by Dr. J. R. Weir, a fungus was observed which upon close study proved to be a new genus. Its morphology places it in *Dematiaceae* under Dictyosporae (Figs. 1 and 2). One conspicuous character of the fungus is a hyaline envelope which envelops the spore. This envelope may be limited to the spore proper or on occasions it may surround the stipe.

The amount of material for study was extremely limited and no inference can be drawn relative to any pathological significance.

COLEODICTYOSPORA

Hyphae scanty, simple or sparingly branched, short, procumbent or ascending, septate, at first hyaline, later colored; conidiophores fasciculate, erect, mostly simple, very occasionally branched, septate, flexuous, delicate, easily separated from the basal mycelium; conidia transversely oblong or inversely reniform, clathrate, clay color to deep olive, surrounded by a semigelatinous, hyaline diaphanous vesicle.

COLEODICTYOSPORA CUBENSIS

Hyphae scanty, procumbent to ascending, septate, hyaline to light fuscous; conidiophores densely fasciculate, slender, mostly simple, irregularly septate, 70–85 x 3.5–5 μ, hyaline, basal portion slightly colored in age, delicate, easily detached from the basal mycelium; conidia transversely oblong or inversely reniform, sometimes slightly constricted, constriction very occasionally under pressure total, separating the conidia into two equal parts, clathrate, tawny-olive, 42–50 x 20–22 μ, vescicle broadly ovate, diaphanous, later disappearing, sometimes enclosing the conidiophores, apical portion rugose-striate on drying, 60–55 x 40–45 μ.

On *Saccharum officinarum*

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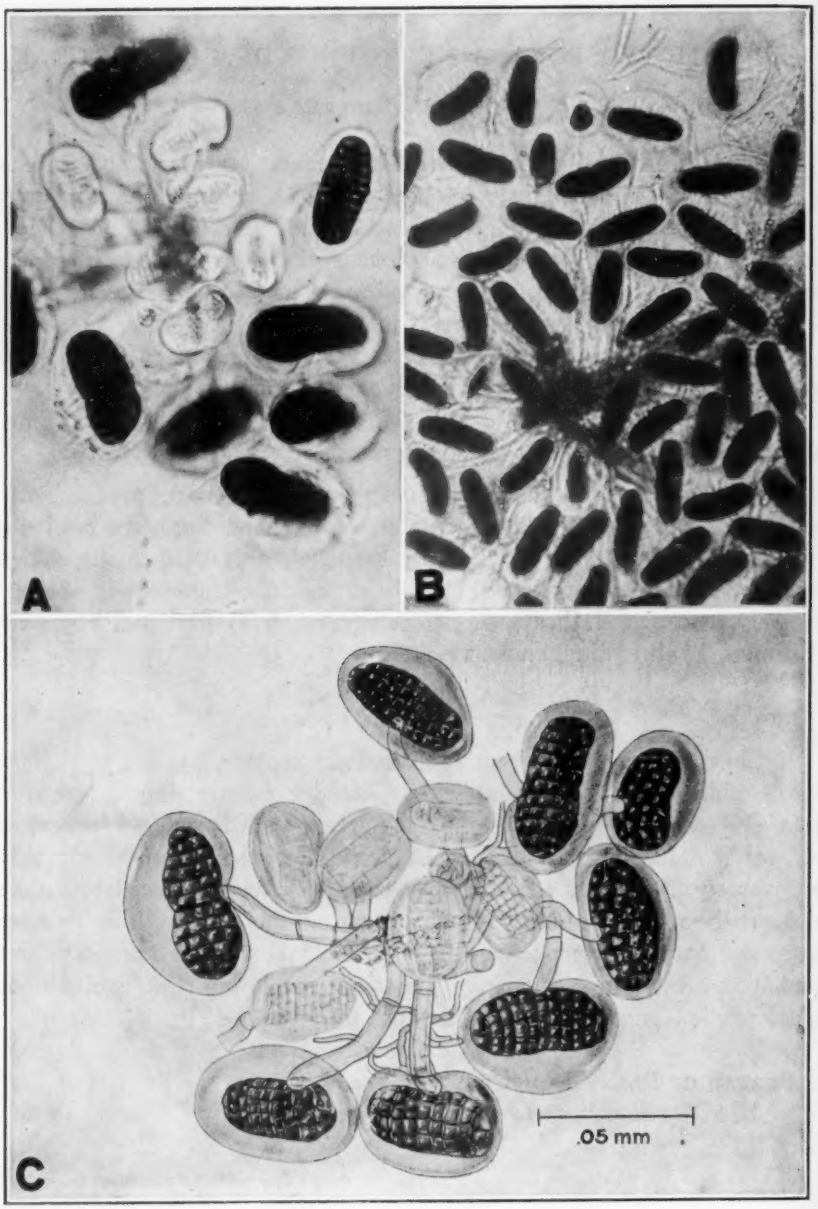

FIG. 1. A. Fascicle of conidiophores showing conidia in different stages of development. B. Fascicle of conidiophores with conidia. C. Interpretative drawing with aid of camera lucida and photograph of surface view of fascicle of conidiophores showing conidia in different stages of development.

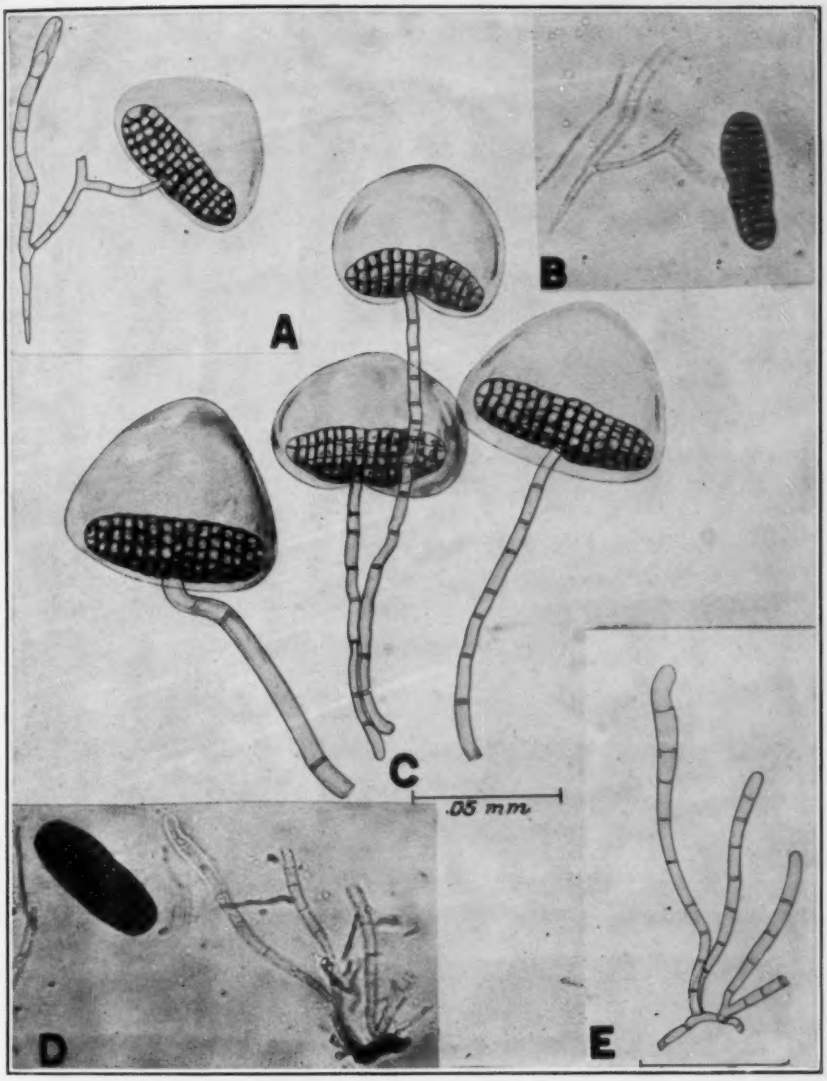

FIG. 2. A. Interpretative drawing with assistance of camera lucida and photograph of single spore showing branched conidiophore. B. Photograph of single spore showing branched conidiophore. C. Individual spores. Interpretative drawing with assistance of camera lucida and photographs. D. Group of conidiophores with conidia detached. E. Interpretative drawing of conidiophores.